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THE TRANSACTIONS

OF THE

EDINBURGH OBSTETRICAL SOCIETY

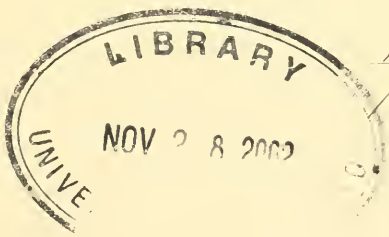
THE TRANSACTIONS

OF THE

EDINBURGH OBSTETRICAL SOCIETY.

VOL. XXXVI.

SESSION 1910-1911.



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PUBLISHERS TO THE SOCIETY.
1911.

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PREFACE.

THIS, the thirty-sixth volume of the Society's Transactions, contains a record of its proceedings during the Session 1910-11.

In it, as in former volumes, the views brought forward in the papers are to be considered as those of the writers themselves, and not as those of the Society as a body.

No meeting of the Society was held in February, as, in place of this, a Complimentary Dinner was given to Dr William Craig on the occasion of his resignation of the office of Treasurer, a post held by him for thirty-five years.

THE EDITOR.

October 1911.

1547

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Sir James Y. Simpson, Bart.,	1866–67	Dr Alexander Ballantyne,	1896–97
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Sir A. R. Simpson,	1882–83		

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	Year.		Year.
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Dr Charles Bell,	1844	Dr Angus Macdonald,	1884
John Kennedy, Esq.,	1844	Dr Charles E. Underhill,	1884–85
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Dr John Moir,	1849–53	Dr A. J. Sinclair,	1889
Dr T. Graham Weir,	1854–55	Sir A. R. Simpson,	1890
Dr John Moir,	1854–55	Dr Peter A. Young,	1890–91
Dr W. Cumming,	1856–57	Dr John Playfair,	1891–92
Dr A. Thomson,	1856–57	Dr Freeland Barbour,	1892–93
Dr T. H. Pattison,	1858–59	Dr A. Ballantyne,	1893–94
Dr David Wilson,	1858–59	Dr James Ritchie,	1894–95
Dr T. Graham Weir,	1860–61	Sir J. Halliday Croom,	1895–96
Dr George S. Keith,	1860–61	Sir A. R. Simpson,	1896–97
Dr T. Graham Weir,	1862–63	Dr R. Milne Murray,	1897–98
Dr Alex. Keiller,	1862–63	Dr N. T. Brewis,	1898–99
Dr T. H. Pattison,	1864–65	Dr J. W. Ballantyne,	1899–1900
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Dr Charles Bell,	1866–67	Dr J. Haig Ferguson,	1902–1903
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Dr L. R. Thomson,	1870–71	Dr D. Berry Hart,	1906–1907
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Dr James Young,	1876–77	Dr N. T. Brewis,	1910
Dr Alex. Milne,	1876–77		

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Dr Cumming, . .	1847 to 1854	Dr William Craig, .	1875 to 1910
Dr Keiller, . . .	1854 to 1859	Dr Barbour Simpson .	1910

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Dr Cumming, . .	1847 to 1854	Dr A. H. Freeland Barbour,	1881 to 1886
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Dr Keiller, . . .	1852 to 1859	Dr J. W. Ballantyne, .	1889 to 1896
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Dr Alexander Milne, .	1873 to 1875		

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Dr Peter Young, . .	1883 to 1889	Dr Lamond Lackie, .	1909

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Dr N. T. Brewis, .	1899 to 1901	Dr W. Macrae Taylor, .	1907 to 1910
Dr J. Lamond Lackie, .	1901 to 1905	Dr Oliphant Nicholson,	1910

LIST OF FELLOWS OF THE SOCIETY.

HONORARY FELLOWS.

1897 Bantock, Dr George Granville, Dunrobin, Payne's Lane, Pinner, Middx.

1901 Bar, Prof. Paul, M.D., Rue la Boëtie, 122, Paris.

1906 Bossi, Professor L. M., The University, Genoa.

1886 Bozeman, Dr Nathan, 296 Fifth Avenue, New York.

1893 Coe, Prof. Henry C., M.D., 27 East Sixty-fourth St., New York.

1910 Döderlein, Professor Albert Sigmund Gustav, Munich.

1898 Doyen, E., M.D., LL.D., Rue Piccini, 6, Paris.

1882 Emmet, Dr, 93 Madison Avenue, New York.

1900 Fehling, Professor Herman, M.D., Kaiser Wilhelm's University, Strassburg.

1882 Freund, Emeritus Professor W., Kleiststrasse, 5, Berlin.

* Previous to 1861 the office of Treasurer was conjoined with that of Senior Secretary.

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1902 Garrigues, Prof. H. J., Tryon, North Carolina.
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1892 Koeberlé, Dr Eugene, Strassburg.
1906 Makičyeff, Professor Alexander Matvějévic, The University, Moscow.
1895 Martin, Emeritus Prof. Dr A., N. of Greifswald, Berlin W., 62 Keithsir 14, vi, 9647.
1903 Morisani, Professor O., San Felice à Piazza Dante, 10, Naples.
1892 Müller, Professor Peter, Berne, Switzerland.
1889 Olshausen, Professor, Frauenklinik, Artillerie Strasse, 13, Berlin.
1901 Ott, Professor D. von, M.D., Professor of Obstetrics, University of St Petersburg.
1902 Pestalozza, Professor, Instituts Obstetrico Policlinico, Roma.
1895 Pinard, Professor A., Rue Cambacérès, 10, Paris.
1898 Pozzi, Professor S., M.D., Hôpital Broca, Paris.
1903 Schauta, Professor, Kochgasse, 16, Vienna.
1882 Schultze, Professor B. S., University, Jena.
1903 Segond, Dr Paul, Quai d'Orsay, Paris.
1906 Simpson, Emeritus Professor Sir Alexander R., LL.D., 52 Queen Street.
1905 Sinclair, Prof. Sir William Japp, Garvock House, Dudley Road, Manchester.
1901 Sneguireff, Professor W., University of Moscow.
1876 Turner, Principal Sir W., K.C.B., LL.D., D.C.L., 6 Eton Terrace, Edinburgh.
1905 Veit, Professor, University, Halle.
1910 Wertheim, Professor Ernst, Vienna.
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1882 Winckel, Prof. Von, Ludwig-Maximilian's University, Munich.
1905 Zweifel, Professor, Frauenklinik University, Leipzig.

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1880 Bosch, Dr Van Den, Liège.
1863 Brown, Dr R. C., Preston.
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1869 Davies, Mr Thos., Sheffield.
1873 Donovan, Mr W., Birmingham.
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1877 Grassett, Dr F., Toronto.
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1897 Günsberg, Charkow, Russia.
1853 Hall, Dr D., Montreal.
1870 Haynes, Dr Stanley L., Malvern.
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1885 Helme, Dr T. A., Manchester.
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1893 Howard-Jones, Dr J., Newport.
1887 Hume, Dr T., Surgeon-Major, India.
1881 Hurst, Dr George, Australia.
1882 Husband, Dr H. Aubrey, Manitoba.
1893 Hutchison, Dr Robert, London.
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1871 Johnston, Dr A. C., R.N., London.
1882 Johnston, Sur.-Maj. Wilson, India.

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1867 Lord, Dr Richard, London.

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1869 M'Millan, Dr T. L., Australia.
1879 Marshall, Dr Thomas, London.
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1887 Shiels, Dr G. F., San Francisco.
1870 Smith, Dr D., Montrose.
1890 Smith, Dr William, America.
1888 Stevenson, Sir Edmond Sinclair, Cape of Good Hope.
1854 Storer, Dr H., Boston, U.S.A.
1875 Sutugin, Dr V., St Petersburg.

1867 Thomson, Mr W., Wrenbury.
1880 Turner, Dr William, Gibraltar.

1885 Underhill, Dr F. T., Vancouver.

1864 Whiteford, Dr James, Greenock.
1886 Whitton, Dr A. B., Aberchirder.
1865 Wollowicz, Dr C., St Petersburg.

ORDINARY FELLOWS.

ARRANGED CHRONOLOGICALLY.

Note.—Those marked with an asterisk have been Members of Council. Members of Council continue in office two years.

		Date of Admission
	*Professor William Stephenson, M.D., F.R.C.S.Ed., *Aberdeen*,	1861
	John Charles Ogilvie Will, M.D., C.M., *Aberdeen*, . .	1867
	*William Taylor, M.D., F.R.C.P. Ed., ...	1868
	Sir James Ormiston Affleck, M.D., LL.D., F.R.C.S. Ed., F.R.C.P. Ed., ...	1869
5	*William Craig, M.D., F.R.C.S. Ed., ...	1870
	****Professor Sir John Halliday Croom, M.D., F.R.C.S. Ed., F.R.C.P. Ed., ...	1870
	**Alexander Ballantyne, M.D., F.R.C.P. Ed., *Dalkeith*, .	1870
	William Borwick Robertson, M.D., L.R.C.S. Ed., *London*, .	1870
	**James Carmichael, M.D., F.R.C.P. Ed., . . .	1871
10	***John Playfair, M.D., F.R.C.P. Ed., Hon. F.R.C.S.Ed., .	1873
	**Henry Macdonald Church, M.D., F.R.C.P. Ed., . .	1875
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	Joshua John Cox, M.D., F.R.C.S. Ed., *Eccles, Manchester*, .	1876
	*Thomas Rutherford Ronaldson, M.B., F.R.C.P. Ed., . .	1876
15	Charles H. Thatcher, F.R.C.S. Ed., ...	1876
	*John Brown Buist, M.D., F.R.C.P. Ed., . . .	1877

		Date of Admission.
	George Herbert Bentley, L.R.C.P. & S. Ed., *Kirkliston*,	1877
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	*****David Berry Hart, M.D., F.R.C.P. Ed.,	1877
20	*David Menzies, M.B., F.R.C.S. Ed.,	1877
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	George Rothwell Adam, M.D., C.M., *Melbourne*,	1879
	*****Alexander Hugh Freeland Barbour, M.D., F.R.C.P. Ed.,	1879
30	James Murray, M.B., C.M.,	1879
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	****James Ritchie, M.D., F.R.C.S. Ed., F.R.C.P. Ed.,	1880
	Thomas Rennie Scott, M.D., C.M., *Musselburgh*,	1880
35	John E. Ranking, M.D., F.R.C.P.L., M.R.C.S. Eng., *Tunbridge Wells*,	1881
	*Arthur Douglas Webster, M.D., F.R.C.P. Ed.,	1881
	James Hewetson, M.B., C.M., *Holmfield, Reigate*,	1881
	**Samuel MacVie, M.B., C.M., *Chirnside*,	1881
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	Henry Anderson Peddie, M.B., C.M.,	1882
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	Andrew Stark Currie, M.D., M.R.C.S. Eng., *London*,	1882
	William Black Alexander, L.R.F.P. & S. Glasg.,	1882
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	David Smart, M.B., C.M., *Liverpool*,	1882
	***Nathaniel Thomas Brewis, M.B., F.R.C.P. Ed., F.R.C.S. Ed.,	1883
	***John William Ballantyne, M.D., F.R.C.P. Ed.,	1883
50	Thomas Proudfoot, M.B., F.R.C.P. Ed.,	1884
	W. Fraser Macdonald, M.B., C.M., *Glasgow*,	1884
	William Wright Millard, M.B., C.M.,	1884
	John Mowat, M.D., C.M.,	1884
	James Lumsden Bell, M.B., C.M., *Driffield, Yorkshire*,	1884
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	*Robert William Felkin, M.D., *London*,	1884
	James K. King, M.D., *Watkins, New York*,	1884
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60	*John Struthers Stewart, L.R.C.P. & S. Ed.,	1884
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	Gustave Michael, M.B., C.M., *London*,	1885
65	***James Haig Ferguson, M.D., F.R.C.P. Ed., M.R.C.S. Eng., F.R.C.S. Ed.,	1885
	John Edward Gemmell, M.B., C.M., *Liverpool*,	1885
	Robert Stewart, M.B., C.M.,	1885
	Surgeon-Captain Robert Charles Macwatt, M.B., B.Sc., C.M., 7th Bengal Cavalry, *Bombay*,	1885
	**E. H. Lawrence Oliphant, M.D., C.M., *Glasgow*,	1885

		Date of Admission.
70	James Hogarth Pringle, M.B., F.R.C.S. Eng., *Glasgow*,	1886
	John Walton Hamp, L.R.F.P. & S. Glasg., L.S.A., *Wolverhampton*,	1886
	James Auriol Armitage, M.D., C.M., *Wolverhampton*,	1886
	*William Henry Miller, M.D., F.R.C.P. Ed.,	1886
	John M'Call, L.R.C.P. Ed., *Portobello*,	1886
75	*Thomas Wood, M.D., C.M.,	1886
	Nutting Stuart Fraser, M.B., M.R.C.S. Eng., *St John's, Newfoundland*,	1887
	Augustus Alexander Matheson, M.D., F.R.C.P. Ed.,	1887
	Robert Mackenzie, M.D., C.M., *Nairn*,	1887
	Thomas Jackson Thyne, M.B., F.R.C.P. Ed.,	1887
80	Ernest T. Roberton, M.D., M.R.C.S. Eng., *New Zealand*,	1887
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85	Arthur Perigal, M.D., M.R.C.S. Eng., *New Barnet, Herts*,	1887
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	Charles Clark Teacher, M.B., C.M., *North Berwick*,	1887
	Robert Inch, M.B., C.M., *Gorebridge*,	1887
	John Orr, M.B., C.M., *Eccles, Lancashire*,	1887
90	*George Owen Carr Mackness, M.D., C.M., *Broughty-Ferry*.	1887
	Francis Joseph Baildon, M.B., C.M., *Southport*,	1887
	Ralph H. Maddox, M.B., M.R.C.S. Eng., I.M.S., *Bengal*,	1887
	James Williamson Martin, M.D., F.R.C.P. Ed., *Dumfries*,	1887
	James Andrew Blair, M.D., C.M., D.Sc., *Newcastle-on-Tyne*,	1887
95	*John Thomson, M.D., F.R.C.P. Ed.,	1887
	Robert Kirk, M.D., F.R.C.S. Ed., *Bathgate*,	1887
	Richard Joseph Tristan, L.R.C.P. & S. Ed., *Retford, Notts*,	1887
	Robert Henry Blaikie, M.D., F.R.C.S. Ed.,	1888
	A. A. Jervis Pereira, M.D., *Delagoa Bay*,	1888
100	Christopher Martin, M.B., F.R.C.S. Eng., *Birmingham*,	1888
	John George Havelock, M.D., C.M., *Montrose*,	1888
	John Pirie, M.B., C.M.,	1888
	James Gibson Graham, M.D., C.M., *Glasgow*,	1888
	Robert Adams Brewis, M.D., C.M., *Dursley*,	1888
105	John Allison, M.D., C.M., *Kettering, Northampton*,	1888
	Archibald Cowan Guthrie, M.B., C.M.,	1888
	Samuel Beatty, M.B., C.M., *Pitlochry*,	1888
	Professor James Chalmers Cameron, M.D., *Montreal*.	1888
	Albert Edward Morison, M.B., F.R.C.S. Ed., M.R.C.S. Eng., *Sunderland*,	1888
110	George H. Temple, M.B., C.M., *Weston-super-Mare*,	1888
	Norman L. Boxill, M.B., C.M., *Barbados*,	1888
	John Hunter Helm, M.B., C.M., *Ratho*,	1888
	George Scott MacGregor, M.D., C.M., *Glasgow*,	1888
	William Sneddon, M.B., C.M., *Cupar-Fife*,	1888
115	Thomas Watts Eden, M.D., F.R.C.P.L., F.R.C.S.E., *London*,	1888
	*William Fordyce, M.D., F.R.C.P. Ed.,	1888
	Charles E. Harvey, M.B., M.R.C.S. Eng., *Sav-la-Mar, Jamaica*,	1889
	*George Pirrie Boddie, M.B., C.M.,	1889
	James F. W. Ross, M.D., *Toronto, Canada*,	1889
120	Hugh Jamieson, M.D., C.M.,	1889
	Prof. John Clarence Webster, M.D., F.R.C.P. Ed., *Chicago*,	1889
	*William George Aitchison Robertson, M.D., F.R.C.P. Ed.,	1889
	William Basil Orr, M.D., C.M.,	1889
	*Edward Farr Armour, M.B., C.M.,	1889

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125	George Wilkinson, M.D., C.M., *Liverpool*,	1889
	**James Lamond Lackie, M.D., F.R.C.P. Ed.,	1889
	James Wilson, M.B., C.M.,	1889
	Archibald Maclean, M.D., C.M., *Kilmarnock*,	1890
	Thomas Dobson Poole, M.D., C.M., *Linthwaite*,	1890
130	Charles Newberry Cobbett, M.D., C.M., *Alberta*,	1890
	Alexander William Gordon Price, M.B., C.M.,	1890
	Hugh Shapter Robinson, M.R.C.S. Eng., L.R.C.P. Ed., *London*,	1890
	*George Matheson Cullen, M.D., C.M.,	1890
	Frederick Albert L. Lockhart, M.B., C.M., *Montreal, Canada*,	1890
135	Edmund Frederick Tanney Price, M.B., C.M.,	1890
	Ernest Theophilus Roberts, M.D., C.M., *Glasgow*,	1890
	Owen Foulkes Evans, M.D., C.M., *Liverpool*,	1890
	James Duncan Farquharson, M.B., C.M., *Newcastle-on-Tyne*,	1890
	Harvey Littlejohn, M.B., F.R.C.S. Ed.,	1890
140	Robert Wise, M.D., C.M., *London*,	1890
	William Russell, M.D., F.R.C.P. Ed.,	1890
	*Michael Dewar, M.D., C.M.,	1891
	Gains T. Smith, M.D. *Moncton, New Brunswick*,	1891
	Robert Thin, M.B., F.R.C.P. Ed.,	1891
145	Alexander Henry Vassie, M.B., C.M., *London*,	1891
	James Harvey, M.D., C.M.,	1891
	Alexander Henderson, M.B., C.M., *Crieff*,	1891
	James Smith, M.D., C.M.,	1891
	George Balfour Marshall, M.D., C.M., *Glasgow*,	1891
150	William Booth, F.R.C.S. Ed.,	1891
	Richard T. Yoe, M.D., *Louisville, Kentucky, U.S.A.*,	1891
	Alexander Bruce Giles, M.D., C.M.,	1891
	Hamilton Graham Langwill, M.D., F.R.C.P. Ed., *Leith*,	1891
	Herbert Ernest Lee, M.B., C.M., *Australia*,	1892
155	Charles Martin, M.B., C.M., *Newton Abbot*,	1892
	William Murray Cairns, M.B., C.M., *Liverpool*,	1892
	Robert Dundas Helm, M.D., C.M., *Carlisle*,	1892
	James Thomas Moore Giffen, F.R.C.S. Ed., *Chester*,	1892
	Frank Dendle, M.B., D.P.H., *Isleworth*,	1892
160	Simson Carstairs Fowler, M.B., C.M., *Juniper Green*,	1892
	*Prof. John Alexander Campbell Kynoch, M.B., F.R.C.P. Ed., *Dundee*,	1892
	Robert Stirling, M.D., C.M., *Perth*,	1892
	William Henry Vickery, F.R.C.S. Eng., L.R.C.P. Lond., *Weston-super-Mare*,	1892
	William Ramsay Smith, M.B., C.M., *Australia*,	1892
165	John Tod, M.B., C.M., *Leith*,	1892
	George Henry Walter Smith, M.D., C.M., *Sydney, Australia*,	1892
	Charles Cromhall Easterbrook, M.D., C.M., F.R.C.P. Ed., *Dumfries*,	1892
	Walter Petrie Simpson, M.B., C.M., *Bathgate*,	1892
	*James Ernest Moorhouse, M.D., C.M., *Stirling*,	1892
170	David George Davidson, M.B., C.M.,	1892
	*Allen Thomson Sloan, M.D., C.M.,	1893
	Robert Balfour Graham, F.R.C.S. Ed., *Leven, Fife*,	1893
	Albert Frederic Rosa, M.D., C.M.,	1893
	George Benjamin Mitchell, M.B., C.M., *Whitby*,	1893
175	John MacRae, M.D., C.M., *Murrayfield*,	1893
	Philip Grierson Borrowman, M.D., C.M.,	1893
	William Herbert Gregory, M.D., C.M., *Beverley, Yorks*,	1893
	James Gibson Cattanach, M.B., F.R.C.P. Ed.,	1893
	*Alexander Maitland Easterbrook, M.B., C.M., *Gorebridge*,	1893

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180	Robert William Roberts, L.R.C.P. & S. Ed., *North Wales*,	1894
	Claude Buchanan Ker, M.D., F.R.C.P. Ed.,	1894
	Charles Alexander Butchart, M.B., C.M.,	1894
	Frederick Maurice Graham, F.R.C.S. Ed., L.R.C.P. Ed.,	1894
	Robert Hoggan, M.B., C.M., *Liberton*,	1894
185	James Livingstone Thompson, M.B., C.M., *Australia*,	1894
	John Stevens, M.D., F.R.C.P. Ed.,	1894
	Sylvanus Glanville Morris, M.D., C.M., *Mardy*,	1894
	David Robertson Dobie, M.D., C.M., *Crieff*,	1894
	Gopal Govind Vatve, M.D., *Bombay*,	1894
190	Robert William Beesley, M.D., C.M., *Bolton*,	1894
	William A. Stephen, M.D., C.M., *Loftus-in-Cleveland*,	1894
	William Edward Fothergill, M.D., C.M., *Manchester*,	1894
	George Sandison Brock, M.D., C.M., *Rome*,	1894
	*John Martin Munro Kerr, M.B., C.M., F.R.F.P.S.G., *Glasgow*,	1894
195	John Montgomery, M.B., C.M., *Birmingham*,	1895
	*Robert Cochrane Buist, M.D., C.M., *Dundee*,	1895
	Robert Thomson Ferguson, M.B., C.M., *Anstruther*,	1895
	Angus Vallance MacGregor, M.D., C.M., *West Hartlepool*,	1895
	John Struthers, M.B., C.M., *Transkei, South Africa*,	1895
200	B. W. Broad, M.B., C.M., *Cardiff*,	1895
	Edwin Hindmarsh, M.B., C.M., F.R.C.S. Ed., *Bengal*,	1895
	Patrick Mackin, M.D., F.R.C.S. Ed., *New Zealand*,	1895
	G. Edgar Helme, M.B., C.M., *Manchester*,	1895
	Percy Theodore Hughes, M.B., C.M., *Broomsgrove*,	1895
205	John Hosack Fraser, M.B., F.R.C.P. Ed., *Bridge of Allan*,	1895
	Stewart Grant Ogilvy, M.B., C.M., *Fauldhouse*,	1895
	Thomas Howard Morgan, M.D., F.R.C.S. Ed., *Queensland, Australia*,	1895
	William Macrae Taylor, M.B., F.R.C.S. Ed.,	1895
	*David James Graham, M.D., F.R.C.P. Ed.,	1895
210	Walter William Chipman, M.D., F.R.C.S. Ed., *Montreal*,	1895
	John Cumming, M.D., F.R.C.S. Ed., F.R.C.P. Ed.,	1896
	Sol Jervois Aarons, M.D., C.M., *London*,	1896
	Robert Beveridge, M.B., C.M., *Leith*,	1896
	John Anderson, M.B., C.M., *Pitlochry*,	1896
215	Robert Gordon M'Kerron, M.B., C.M., *Aberdeen*,	1896
	Frederick John M'Cann, M.D., F.R.C.S. Eng., *London*,	1896
	David Robert Taylor, L.R.C.P. & S. Ed., *Ayton*,	1896
	George William Simla Paterson, M.B., C.M.,	1896
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220	Thomas Marshall Callender, M.D., C.M., *Sidcup*,	1896
	Lewis Grant, M.D., C.M., *Neston*,	1896
	Robert Robertson, M.B., C.M.,	1897
	James Wilkie, L.R.C.P. & S. Ed., *Portobello*,	1897
	Andrew Graham, M.D., *Currie*,	1897
225	Roderick Murdoch Matheson, M.D., F.R.C.S. Ed.,	1897
	*Robert Jardine, M.D., F.R.F.P.S. Glasg., M.R.C.S. Eng., *Glasgow*,	1897
	Daniel Charles Edington, M.D., C.M., *Penrith*,	1897
	John Macmillan, M.D., F.R.C.P. Ed., F.R.C.S. Ed.,	1897
	Harold Sherman Ballantyne, M.B., F.R.C.P. Ed., *Dalkeith*,	1897
230	Ernest Edward Porritt, M.D., F.R.C.S. Ed., *New Zealand*,	1897
	William John Garbutt, M.B., C.M., *Birmingham*,	1897
	Henry John Forbes Simson, M.B., F.R.C.S. Ed., *London*,	1897
	Angus Macdonald, M.B., F.R.C.S. Ed.,	1897
	George Robert Livingston, M.D., C.M., *Dumfries*,	1898
235	William Morrison Milne, M.B., C.M.,	1898
	William Joseph Murphy Barry, M.D., M.R.C.P. Ed., *Penarth*,	1898

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	Alexander Cruikshank Ainslie, M.D., C.M.,	1898
	Henry Aylmer Dumat, M.D., F.R.C.P.Ed., *Durban, South Africa*,	1898
	John Thomas Woodside, L.R.C.P. & S. Ed., *Glamorganshire*,	1898
240	*George Freeland Barbour Simpson, M.D., F.R.C.S. Ed., F.R.C.P. Ed.,	1898
	Alfred Charles Sandstein, M.D., Ch.B., *New Zealand*,	1898
	Alfred Shearer, M.B., Ch.B., *Newtown, N. Wales*,	1898
	James Duncan Slight, M.D., Ch.B., *Leicester*,	1898
	Francis John Harvey Bateman, M.D., C.M., *London*,	1898
245	Robert John Johnston, M.B., C.M.,	1899
	William Bertie Mackay, M.D., *Berwick-on-Tweed*,	1899
	Edward William Scott Carmichael, M.D., F.R.C.S. Ed.,	1899
	James Wilson M'Brearty, F.R.C.S.Ed., L.R.C.P. Ed., *New Zealand*,	1899
	George Crewdson Thomas, M.D., C.M., *London*,	1899
250	William John Barclay, M.D., F.R.C.S. Ed., *New Zealand*,	1899
	Alexander Dingwell Fordyce, M.D., F.R.C.P. Ed.,	1899
	William Thomas Ritchie, M.D., F.R.C.P. Ed.,	1899
	Owen, St John Moses, M.D., C.M., B.Sc., *India*,	1900
	*Harry Oliphant Nicholson, M.D., F.R.C.P. Ed.,	1900
255	Thomas Scott Brodie, M.B., C.M., *Wishaw*,	1900
	William Hope Fowler, M.B., Ch.B.,	1900
	Ogden Watson Ogden, M.D., M.R.C.S., *Newcastle-on-Tyne*,	1900
	John Craig, M.B., Ch.B.,	1900
	William Hartley Bunting, M.D., F.R.C.S. Ed., *Birmingham*,	1900
260	Theodore Charles Mackenzie, M.B., Ch.B., *Inverness*,	1900
	Hugh Corbett Taylor Young, M.D., C.M., *Sydney*,	1900
	John Boyd Jamieson, M.D., F.R.C.S. Ed.,	1900
	Malcolm M'Larty, M.B., C.M.,	1900
	Frederick Gardiner, M.D., C.M.,	1900
265	George Mackie, M.B., Ch.B., *Malvern*,	1900
	*Kenmure Duncan Melville, M.D., Ch.B.,	1900
	John Thomas Dickie, L.R.C.P. & S. Ed.,	1900
	*William Ernest Frost, M.B., Ch.B.,	1900
	*Frederick David Simpson, M.D., F.R.C.S. Ed.,	1900
270	Francis Wilfrid Harlin, F.R.C.S. Ed., L.R.C.P. Ed., *Queensland*,	1900
	William Darling, M.B., F.R.C.S. Ed.,	1900
	Malcolm Campbell, M.B., F.R.C.S. Ed., *Brechin*,	1900
	James Ramsay Munro, M.D., Ch.B., *Spalding*,	1901
	George James Rogerson Carruthers, M.B., Ch.B.,	1901
275	Hilda Maud M'Farlane Sturrock, L.R.C.P. & S. Ed., *Perth*,	1901
	George Dickson, M.D., C.M.,	1901
	Elsie Maud Inglis, M.B., C.M.,	1901
	George Robertson, L.R.C.P. & S. Ed., *Dunfermline*,	1901
	John Wishart Kerr, M.B., Ch.B., *Glasgow*,	1901
280	William Harold Graham Aspland, M.D., M.R.C.S. Eng., *China*,	1901
	William Taylor McArthur, M.D., F.R.C.S. Ed., *California*,	1901
	Henry Overton Hobson, M.D., C.M., *London*,	1901
	*Robert Patton Ranken Lyle, M.D., Ch.B., *Newcastle-on-Tyne*.	1901
	David Whiteside Maclagan, M.B., Ch.B., *New Zealand*,	1901
285	Kennedy C. M'Ilwraith, M.B., M.C.P. & S. Ont., *Toronto*,	1901
	Robert Alexander John Harper, M.D., Ch.B., *Dalkeith*,	1901
	William Hogg Prentice, M.D., Ch.B., *Pendleton*,	1901
	Alexander Mowatt Malcolmson, M.D., Ch.B., *Corstorphine*,	1901

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	Caleb Williams Saleeby, M.D., Ch.B., *London*,	1902
290	John Ligertwood Green, M.D., Ch.B.,	1902
	William Sloss, M.B., Ch.B., *Australia*,	1902
	Charles James Hill Aitken, M.D., C.M., *Cape Colony*,	1902
	E. R. Secord, M.D., *Ontario*,	1902
	F. E. Thompson, M.D., *Montreal*,	1902
295	John M'Gibbon, M.B., C.M.,	1902
	Thomas James Thomson, M.D., C.M.,	1902
	Charles Mowbray Pearson, M.B., F.R.C.P.Ed.,	1902
	Ewen John Maclean, M.D., M.R.C.P. Lond., *Cardiff*,	1902
	James William Somerville, M.D., C.M., *Galashiels*,	1902
300	Alexander Miller, L.R.C.P. Ed., L.F.P.S. Glasg., *Glasgow*,	1902
	Duncan Macnab Callender, M.B., Ch.B., *Lancaster*,	1902
	Robert Cranston Low, M.B., F.R.C.P.Ed.,	1902
	Benjamin Philp Watson, M.D., F.R.C.S.Ed.,	1902
	John Macdonald, M.B., C.M., *Cupar-Fife*,	1902
305	John Sullivan, M.B., Ch.B.,	1902
	Charles William Somerville, M.B., Ch.B., *China*,	1902
	Frank Mayes Willcox, M.B., C.M.,	1902
	John Tennant, M.B., C.M., *Scunthorpe*,	1902
	Alexander Simpson Wells, M.B., F.R.C.S. Ed., *Cape Town*,	1903
310	Andrew Binny Flett, M.B., Ch.B.,	1903
	William Henry Eden Brand, F.R.C.S. Ed., L.R.C.P. Ed., *Banchory*,	1903
	Francis Cavanagh, M.B., Ch.B., *Sheffield*,	1903
	Alfred Lambré White, L.R.C.P. & S. Ed., *Manchester*,	1903
	Robert Bathgate Johnston, L.R.C.P. & S. Ed., *Penrith*,	1903
315	William Llewellyn Jones, M.D., F.R.C.S. Ed., *Merthyr-Tydvil*,	1903
	Robert Wilson Gibson, M.D., F.R.C.S. Ed., *Orton*,	1903
	Philip Henry Mules, M.B., Ch.B., *New Zealand*,	1903
	Andrea Francis Honyman Rabagliati, M.D., Ch.B., *Bradford*,	1903
	Donald Gregor MacArthur, M.D., C.M., *Aberfeldy*,	1903
320	Henry Martyn Stumbles, M.D., Ch.B., *Amble*,	1903
	Gilbert John Farie, M.B., Ch.B., *London*,	1903
	Katherine Jane Stark Clark, M.D., Ch.B., D.P.H.,	1903
	David Halliday Croom, M.D., Ch.B.,	1903
	Eleanor Russell Elder, M.B., Ch.B., *Leith*,	1903
325	Robert William Johnstone, M.D., Ch.B.,	1903
	James William Keay, M.D., Ch.B.,	1903
	Ivan Cochrane Keir, M.D., Ch.B., *Melksham*,	1903
	Alexander Grant Macdonald, M.B., C.M., *Isle of Skye*,	1903
	Charles John Shaw, M.D., Ch.B., *Lochgilphead*,	1903
330	*Frederick Porter, M.B., C.M.,	1904
	Sherwin Gibbons, M.D., *Los Angeles*,	1904
	Russell Gerald William Adams, M.D., Ch.B., *New Zealand*,	1904
	Duncan Campbell Lloyd Fitzwilliams, M.D., F.R.C.S.Ed., *London*,	1904
	Clarence Brian Dobell, M.B., M.R.C.S. Eng., M.R.C.P.L., *Cheltenham*,	1904
335	Hugh Stevenson Davidson, M.B., Ch.B.,	1904
	Andrew Milroy Fleming, C.M.G., M.B., F.R.C.S. Ed., *Rhodesia*,	1904
	Thomas William Edmonston Ross, M.B., Ch.B., *Cardiff*,	1904
	John Benjamin Hellier, M.D., M.R.C.S. Eng., *Leeds*,	1904
	John Thomas Williams, M.D., *Rhondda Valley*,	1904
340	William Brown, M.B., Ch.B., *Braemar*,	1904

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	John Hepburn Lyell, M.D., C.M., *Perth*,	1904
	Henry Howard Robarts, M.D., Ch.B., *Haddington*,	1904
	Robert Balfour Barnetson, M.B., Ch.B., *Portobello*,	1904
	James Lochhead, M.D., F.R.C.S.Ed.,	1904
345	Arthur Charles Strain, M.D., Ch.B., *West Hartlepool*,	1904
	Lewis Beesly, L.R.C.P.Ed., F.R.C.S. Ed.,	1904
	James Crawford Gibb Macnab, M.B., F.R.C.S. Ed.,	1904
	Robert William Lessel Wallace, M.B., Ch.B., *Egypt*,	1905
	Alfred Thom Bulkeley Gavin, M.B., C.M., *London*,	1905
350	Alastair MacGregor, M.D., C.M., *Greenhithe*,	1905
	Edmond Frost, M.D., C.M., *Eastbourne*,	1905
	Edith Cochrane-Brown Pitts, M.B., Ch.B., *New Zealand*,	1905
	James Brownlee, M.D., Ch.B., *Middlesbrough*,	1905
	Peter M'Ewan, M.B., Ch.B., *Scarborough*,	1905
355	George Douglas Mathewson, M.B., Ch.B.,	1905
	Richard James Harley, M.D., L.R.C.P. & S.Ed., *Blackburn*,	1905
	Andrew Fleming, M.D., Ch.B., *Corstorphine*,	1905
	Robert William Craig, M.D., Ch.B., *Ford*,	1905
	Henry John Dunbar, M.D., Ch.B.,	1905
360	Richard Alfred Blake, M.D., Ch.B., *Transvaal*,	1905
	John Herbert Gibbs, F.R.C.S.Ed.,	1905
	Alice Marion Hutchison, M.D., Ch.B.,	1905
	W. T. Chenhall, M.D., *Sydney*,	1906
	Alexander Angus Martin, F.R.C.S.Ed., *North Shields*,	1906
365	James Andrew Gunn, M.D., Ch.B.,	1906
	James Lawson Russell, M.B., Ch.B., *Todmorden*,	1906
	Archibald M'Kendrick, F.R.C.S. Ed., L.R.C.P. Ed.,	1906
	Alexander Scott, M.B., C.M., *Broxburn*,	1906
	Archibald Simpson, M.B., Ch.B., *Tranent*,	1906
370	Hirjee Nowroje Anklesaria, L.R.C.P. & S.Ed., *Bombay*,	1906
	Archibald Cotterell M'Master, M.B., Ch.B., *New Zealand*,	1906
	Herbert Pank Thompson, M.D., F.R.C.S. Ed., *Harrogate*,	1906
	Samuel Davidson, M.D., C.M., *Kelso*,	1906
	William Fowler Godfrey, M.B., C.M., *Manitoba*,	1906
375	Kaikhuson Dadabhoy, F.R.C.S.Ed., L.R.C.P.Ed., *India*,	1906
	Edward Burnet, M.B., Ch.B., *Leamington*,	1906
	Arthur Samuel Walker, M.B., Ch.B., *Leith*,	1906
	Archibald Dunlop Stewart, M.B., L.R.C.S. Ed.,	1906
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380	Ethelbert William Dyer, M.B., F.R.C.S.Ed., *Durban*,	1906
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	John Bruce M'Moreland, M.B., Ch.B.,	1906
	William Omand Sclater, M.B., Ch.B., *Jarrow-on-Tyne*,	1906
385	Archibald George Kirkwood Ledger, M.D., Ch.B., *Coppull*,	1906
	Frederick James Greig, L.R.C.P. & S.I., Lt.-Col., R.A.M.C., *Stirling*,	1906
	Duncan Lorimer, M.B., F.R.C.S.Ed.,	1906
	Charles Robert Paterson Mitchell, M.B., Ch.B., *Malvern*,	1906
	John Halley Meikle, M.D., D.P.H.,	1906
390	David Lloyd Roberts, M.D., F.R.C.P., *Manchester*,	1906
	Elsie Mary Macmillan Barnetson, M.B., Ch.B., *Joppa*,	1907
	John Andrew Macleod, M.B., Ch.B., *Inverness*,	1907
	Hugh Smith Reid, M.D., F.R.C.S.Ed.,	1907
	Allan Macdonald Dick, M.B., Ch.B., I.M.S., F.R.C.S.Eng., L.R.C.P.Lond., *Bombay*,	1907
395	Angus Buchanan, M.B., Ch.B.,	1907
	William Bow Tannahill, M.B., Ch.B., *Greenock*,	1907

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	Isabella Beattie Venters, M.B., Ch.B.,	1907
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400	Ronald Græme Scott Orbell, M.D., Ch.B., *New Zealand*,	1907
	John Alexander Gray, M.B., Ch.B.,	1907
	Cecil Hobart Houghton, M.B., Ch.B., *Cape Colony*,	1907
	Robert Patrick M'Morland, M.B., Ch.B.,	1907
	James Young, M.D., F.R.C.S.Ed.,	1907
405	John Nigel Stark, M.D., F.R.F.P.S.G., *Glasgow*,	1908
	Norman Scott Carmichael, M.B., Ch.B.,	1908
	Harley Patterson Milligan, M.D., F.R.C.S.Ed., *Hessle*,	1908
	Thomas Yule Finlay, M.B., Ch.B.,	1908
	David Lawrence Luckhoo, M.D., L.R.C.P.S.Ed., *British Guiana*,	1908
410	James Crawford Craig, M.B., Ch.B., *Aylesbury*,	1908
	John Gibson Craig, M.B., F.R.C.S.Ed., *York*,	1908
	Alexander Gibson, M.B., Ch.B.,	1908
	Hugh Ruthven Lawrence, M.D., Ch.B.,	1908
	Alexander Cross Mallace, M.B., Ch.B.,	1908
415	John More, L.R.C.P.Lond., M.R.C.S.Eng., *Rothwell*,	1908
	Percy Wilfred Shepherd, M.B., C.M., *Leeds*,	1908
	David Elliot Dickson, M.D., F.R.C.S.Ed., *Lochgelly*,	1908
	John M'Culloch, M.D., C.M., *Ontario*,	1909
	Robert Brodie Anderson, M.D., F.R.C.S.Ed., *Manitoba*,	1909
420	George Scott Carmichael, M.D., Ch.B.,	1909
	William James Crow, M.B., Ch.B.,	1909
	Frank Porter Patterson, M.D., F.R.C.S.Ed., *Canada*,	1909
	Alfred Edward Turnbull, M.B., Ch.B., *Dunbar*,	1909
	Alexander Aitken Ross, M.B., Ch.B.,	1910
425	Stephen Forrest, M.D., Ch.B.,	1910
	Herbert Lindesay Watson Wemyss, M.D., Ch.B., *Broughty-Ferry*,	1910
	John M'Laren, M.B., C.M.,	1910
	William Fraser Macdonald, M.B., Ch.B.,	1910
	Morgan Howard Howard-Jones, L.R.C.P. & S.Ed.,	1910
430	R. C. Alexander, M.B., Ch.B.,	1910
	J. Keith B. MacGlashan, M.B., Ch.B.,	1910
	Matthew R. Drennan, M.A., M.B., Ch.B.,	1910
	F. E. Jardine, M.B., Ch.B., *Penicuik*,	1910
	R. S. Mowat, M.D.	1910
435	W. W. Carlow, M.B., Ch.B.,	1910
	J. M. Dewar, M.B., Ch.B.,	1910
	A. J. Gordon Hunter, M.B., Ch.B., *Linlithgow*	1910
	W. Menzies Menzies, M.B., Ch.B.,	1910
	W. P. Murray, M.B., Ch.B.,	1910
440	David Shannon, M.B., Ch.B., *Glasgow*,	1910
	Lewis H. F. Thatcher, M.D., M.R.C.P.E.,	1910
	A. L. S. Tuke, M.B., C.M., *Dunfermline*,	1911
443	James Bruce Ronaldson, M.R.C.S., L.R.C.P., *London*,	1911

ORDINARY FELLOWS.

ARRANGED ALPHABETICALLY.

(*a.*) LIFE MEMBERS.

		Date of Admission.
	Aarons, Dr S. Jervois, 6 Hartley Street, Cavendish Square, London, W.,	1896
	Adam, Dr George Rothwell, 84 Collins Street, Melbourne, Australia,	1879
	Anderson, Dr John, Newholme, Pitlochry,	1896
	Ballantyne, Dr Harold S., Ashton, Eskbank, Dalkeith,	1897
5	Ballantyne, Dr J. W., 19 Rothesay Terrace,	1883
	Barbour, Dr A. H. Freeland, 4 Charlotte Square,	1879
	Barclay, Dr William John, Invercargill, New Zealand,	1899
	Barrington, Dr Fourness, 213 Macquarrie Street, Sydney, Australia,	1884
	Brock, Dr G. Sandison, 6 Corso d'Italia, Rome,	1894
10	Burnet, Dr Edward, Dalblair, Lillington Road, Leamington,	1906
	Cavanagh, Dr Francis, 396 Ecclesall Road, Sheffield,	1903
	Chenhall, Dr William T., 233 Macquarie St., Sydney, Australia,	1906
	Chipman, Dr W. W., 285 Mountain St., Montreal, Canada,	1895
	Craig, Dr John, 71 Bruntsfield Place,	1900
15	Craig, Dr William, 71 Bruntsfield Place,	1870
	Croom, Dr David Halliday, 17 Alva St.,	1903
	Croom, Prof. Sir John Halliday, 25 Charlotte Square,	1870
	Cumming, Dr John, 70 Bruntsfield Place,	1896
	Dobell, Dr C. B., 1 Royal Well Terrace, Charlton, Cheltenham,	1904
20	Dumat, Dr Henry Aylmer, 7 Devonshire Place, Durban, Natal, South Africa,	1898
	Ferguson, Dr J. Haig, 7 Coates Crescent,	1885
	Fitzwilliams, Dr Gerald Lloyd, Cilgwyn, N.C. Emlyn, R.S.O., Carmarthenshire,	1907
	Fleming, Dr Andrew M., C.M.G., Salisbury, Rhodesia,	1904
	Fowler, Dr Simson, Waverley, Juniper Green,	1892
25	Frost, Dr Edmund, Chesterfield, Meads, Eastbourne,	1905
	Gibson, Dr R. Wilson, Town Head House, Orton, Tebay,	1903
	Grant, Dr Lewis, Neston, Cheshire,	1896
	Hart, Dr D. Berry, 5 Randolph Cliff,	1877
	Haultain, Dr F. W. N., 12 Charlotte Square,	1884
30	Houghton, Dr C. Hobart, Fort Hare, Alice, Cape Colony, South Africa,	1907
	Inch, Dr Robert, Gorebridge,	1887
	Livingston, Dr George R., 47 Castle Street, Dumfries,	1898
	Luckhoo, Dr David Lawrence, New Amsterdam, British Guiana,	1908
	M'Arthur, Dr W. Taylor, 2025 Western Avenue, Los Angeles, California,	1901
35	M'Brearty, Dr J. Wilson, Greymouth, West Coast, New Zealand,	1899
	M'Culloch, Dr John. Lindsay, Ontario, Canada,	1909
	Macnab, Dr James C. G., Germiston, Transvaal, South Africa	1904
	Maddox, Dr Ralph H., I.M.S., c/o Messrs Thomas Cook & Son, Ludgate Circus, London, E.C.,	1887
	Martin, Dr Christopher, Cleveland House, George Road, Edgbaston, Birmingham,	1888
40	Melville, Dr Kenmure, 2 Nile Grove,	1900

		Date of Admission.
	Morgan, Dr T. H., Gympie, Queensland, Australia,	1895
	Mules, Dr P. Henry, Bishopdale, Nelson, New Zealand,	1903
	Pereira, Dr A. A. Jervis, Physician to the Mozambique Company, Beira, East Africa,	1888
	Pitts, Dr Edith Cochrane-Brown, Strathmore, Christ Church, New Zealand,	1905
45	Ranking, Dr J. E., Tunbridge Wells,	1881
	Ross, Dr James F. W., 481 Sherbourne Street, Toronto, Canada,	1889
	Russell, Dr J. Lawson, West Lodge, Tormorden,	1906
	Simpson, Dr G. F. Barbour, 43 Manor Place,	1898
	Simpson, Dr W. Petrie, Viewbank, Bathgate,	1892
50	Simson, Dr H. J. F., 36 Grosvenor Street, London, W.,	1897
	Sloss, Dr William, Sturt Street, Ballarat, Australia,	1902
	Struthers, Dr John, Nqamakwe, Transkei, South Africa,	1895
	Sturrock, Dr Hilda M., Northcote, Edinburgh Road, Perth,	1901
	Vatve, Dr Gopal Govind, c/o H.H. The Rajah of Miraj, Bombay, India,	1894
55	Wells, Dr A. Simpson, 118 New Church Street, Cape Town, South Africa,	1903

(*b.*) ANNUAL SUBSCRIBERS.

	Adams, Dr Russell G. W., Maxwell Road, Blenheim, New Zealand,	1904
	Allleck, Sir J. O., 38 Heriot Row,	1869
	Ainslie, Dr A. C., 49 Minto Street,	1898
	Aitken, Dr C. J. Hill, Idutywa, Transkei, Cape Colony, South Africa,	1902
60	Alexander, Dr R. C., 15 Hartington Gardens,	1910
	Alexander, Dr W. B., 8 Blenheim Place,	1882
	Allison, Dr J., Fuller House, Kettering, Northampton,	1888
	Anderson, Dr R. Brodie, F.R.C.S.Ed., 366 Main Street, Winnipeg, Manitoba, Canada,	1909
	Anklesaria, Dr H. N., Taj Building, Hornby Road, Bombay, India,	1906
65	Armitage, Dr J. A., 58 Waterloo Road, Wolverhampton,	1886
	Armour, Dr E. F., 6 Bruntsfield Terrace,	1889
	Aspland, Dr W. H. Graham, Church of England Mission, Peking, China,	1901
	Baildon, Dr Francis J., 42 Hoghton Street, Southport,	1887
	Ballantyne, Dr A., Ashton, Eskbank, Dalkeith,	1870
70	Barnetson, Dr Elsie M., Woodville, Joppa,	1907
	Barnetson, Dr R. Balfour, Woodville, Joppa,	1904
	Barry, Dr W. J. M., 29 Plymouth Road, Penarth, Glamorgan,	1898
	Bateman, Dr F. J. Harvey, Heath End, Blackheath, Lond., S.E.,	1898
	Beatty, Dr Samuel, Craigvar, Pitlochry,	1888
75	Beesley, Dr R. W., 135 Deane Road, Bolton,	1894
	Beesly, Dr Lewis, 40 Northumberland Street,	1904
	Bell, Dr J. Lumsden, The White Hall, Driffield, Yorkshire,	1884
	Bentley, Dr G. H., Loanhead House, Kirkliston,	1877
	Beveridge, Dr Robert, 9 James Place, Leith,	1896
80	Blaikie, Dr R. H., 10 Mayfield Gardens,	1888
	Blair, Dr J. A., 16 Windsor Terrace, Newcastle-on-Tyne,	1887
	Blake, Dr R. A., Belfast, Transvaal, South Africa,	1905
	Boddie, Dr G. P., 35 Melville Street,	1889
	Booth, Dr William, 2 Minto Street,	1891

		Date of Admission
85	Borrowman, Dr Philip G., 21 Pitt Street,	1893
	Boxill, Dr N. L., Buttalls, St George, Barbados,	1888
	Brand, Dr Eden, Bellfield, Banchory,	1903
	Brewis, Dr N. T., 6 Drumsheugh Gardens,	1883
	Brewis, Dr R. Adams, The West Gate, Dursley, Gloucestershire,	1888
90	Broad, Dr B. W., The Sanitorium, Cardiff,	1895
	Brodie, Dr T. Scott, 21 Belhaven Terrace, Wishaw,	1900
	Brown, Dr William, Auchendryne Lodge, Braemar,	1904
	Brownlee, Dr James, Waterloo House, Linthorpe Road, Middlesbrough,	1905
	Buchanan, Dr Angus, 3 Whitehouse Loan,	1907
95	Buist, Dr J. W., 1 Clifton Terrace,	1877
	Buist, Dr R. C., 166 Nethergate, Dundee,	1895
	Bunting, Dr W. Hartley, 20 Hagley Road, Edgbaston, Birmingham,	1900
	Butchart, Dr C. A., 52 Leith Walk, Leith,	1894
	Cairns, Dr W. Murray, 67 Catharine Street, Liverpool,	1892
100	Calder, Dr H. L., 60 Leith Walk, Leith,	1882
	Callender, Dr D. M., 6 Rose Bank, Lancaster,	1902
	Callender, Dr T. M., Inverard, Sidcup,	1896
	Cameron, Prof. James C., M.D., 605 Dorchester Street, W., Montreal,	1888
	Campbell, Dr Malcolm, Westwood, Brechin,	1900
105	Carlow, Dr W. W., 20 Eglinton Crescent,	1910
	Carmichael, Dr Edward, 21 Abercromby Place,	1887
	Carmichael, Dr E. W. Scott, 40 Palmerston Place,	1899
	Carmichael, Dr George S., 25 Braid Road,	1909
	Carmichael, Dr James, 22 Northumberland Street,	1871
110	Carmichael, Dr Norman Scott, 22 Northumberland Street,	1908
	Carruthers, Dr G. J. R., 4 Melville Street,	1901
	Cattanach, Dr J. G., 3 Alvanley Terrace,	1893
	Church, Dr H. M., 36 George Square,	1875
	Clark, Dr Katherine S., 16 Drummond Place,	1903
115	Cobbett, Dr C. N., 632 Fourth Street, Edmonton, Alberta, Canada,	1890
	Cox, Dr Joshua J., 38 Deansgate, Manchester.	1876
	Craig, Dr James C., Melrose. Aylesbury, Bucks,	1908
	Craig, Dr J. G., 78 Bootham, York,	1908
	Craig, Dr R. W., Pathhead-Ford, Dalkeith,	1905
120	Croudace, Dr J. H., Foregate House, Stafford,	1878
	Crow, Dr W. J., 108 Morningside Road,	1909
	Cullen, Dr G. M., 50 Minto Street,	1890
	Currie, Dr A. S., 20 Oxford Terrace, Hyde Park, London, W.,	1882
	Dadabhoy, Dr K., Victoria Road, Karachi, India,	1906
125	Darling, Dr T. Brown, 13 Merchiston Place,	1884
	Darling, Dr William, 2 Warrender Park Terrace,	1900
	Davidson, Dr D. G., 9 Granville Terrace,	1892
	Davidson, Dr H. S., 4 Dundas Street,	1904
	Davidson, Dr Samuel, Mansefield, Kelso,	1906
130	Davies, Dr Arnold, 5 Ruthin Gardens, Cathays, Cardiff, North Wales,	1906
	Dendle, Dr Frank, Overton House, Spring Grove, Isleworth,	1892
	Dewar, Dr J. M., 24 Lauriston Place,	1910
	Dewar, Dr M., 24 Lauriston Place,	1891
	Dick, Dr A. M., c/o Grindlay Grome Co., Bombay, India,	1907
135	Dickie, Dr J. T., 37 Lauriston Place,	1900
	Dickson, Dr D. Elliot, Hillcrest, Lochgelly,	1908
	Dickson, Dr George, 26 Merchiston Park,	1901
	Dobie, Dr D. Robertson, 7 James Square, Crieff,	1894

		Date of Admission.
	Drennan, Dr Matthew R., The Residency, Edinburgh Royal Infirmary,	1910
140	Dunbar, Dr H. J., 3 Hampton Terrace,	1905
	Dyer, Dr Ethelbert W., Lustleigh, 132 Davenport Road, Durban, South Africa,	1906
	Easterbrook, Dr A. M., Arnprior, Gorebridge,	1893
	Easterbrook, Dr C. C., Crichton House, Dumfries,	1892
	Eden, Dr T. Watts, 26 Queen Anne Street, Cavendish Square, London, W.,	1888
145	Edington, Dr D. C., Birbeck House, Penrith,	1897
	Elder, Dr Edward A., 6 Torphichen Street,	1906
	Elder, Dr Eleanor, 4 John's Place, Leith,	1903
	Elder, Dr W. Nicol, 6 Torphichen Street,	1879
	Evans, Dr O. F., 20 Princes Avenue, Liverpool,	1890
150	Farie, Dr G. J., Greville House, Raynes Park, Wimbledon, London, S.W.,	1903
	Farquharson, Dr J. D., 242 Westgate Road, Newcastle-on-Tyne,	1890
	Felkin, Dr R. W., 47 Bassett Road, North Kensington, London, W.,	1884
	Ferguson, Dr R. T., Middlemarch, Anstruther, Fife,	1895
	Finlay, Dr T. Y., 17 Drummond Place,	1908
155	Fitzwilliams, Dr Duncan C. L., 31 Grosvenor Street, Grosvenor Square, London, W.,	1904
	Fleming, Dr Andrew, St John's Road, Corstorphine,	1905
	Flett, Dr A. B., 60 George Square,	1903
	Fordyce, Dr A. Dingwall, 8 Manor Place,	1899
	Fordyce, Dr William, 20 Charlotte Square,	1888
160	Forrest, Dr Stephen, 1 Magdala Crescent,	1910
	Fothergill, Dr W. Edward, 337 Oxford Road, Manchester,	1894
	Fowler, Dr W. Hope, 21 Walker Street,	1900
	Fox, Dr J. W., 18 Bernard Street, Southampton,	1887
	Fraser, Dr J. Hossack, Fernfield, Bridge of Allan,	1895
165	Fraser, Dr Nutting S., 205 Gower Street, St John's, Newfoundland,	1887
	Frost, Dr W. E., 36 Moray Place,	1900
	Garbutt, Dr W. J., Girthen House, Bournbrook Rd., Selly Pk., Birmingham,	1897
	Gardiner, Dr Frederick, 9 George Square,	1900
	Gavin, Dr A. T. Bulkeley, 8 Pont Street, Belgrave Square, London, S.W.,	1905
170	Gayton, Dr William, Ravensworth, Regent's Park Road, Finchley, London, N.,	1885
	Gemmell, Dr J. E., 28 Rodney Street, Liverpool,	1885
	Gibbons, Dr Sherwin, 1010 Union Trust Building, Los Angeles, California,	1904
	Gibbs, Dr J. H., 12 Coates Crescent,	1905
	Gibson, Dr Alexander, 14 Scotland Street,	1908
175	Giffen, Dr J. T. M., 138 Boughton, Chester,	1892
	Giles, Dr A. B., 4 Palmerston Place,	1891
	Gilmour, Dr T. E., Port Ellen, Islay,	1882
	Godfrey, Dr W. F., Carberry, Manitoba, Canada,	1906
	Graham, Dr A., Curriebank, Currie,	1897
180	Graham, Dr D. J., 26 Rutland Street,	1895
	Graham, Dr F. M., 16 Mayfield Gardens,	1894
	Graham, Dr J. Gibson, 17 Ashton Ter., Dowanhill, Glasgow,	1888
	Graham, Dr R. Balfour, Leven, Fife,	1893
	Gray, Dr J. A., 4 Wolseley Terrace,	1907
185	Green, Dr John Ligertwood, 23 Minto Street,	1902

		Date of Admission.
	Gregory, Dr W. H., North Bar Without, Beverley, Yorks,	1893
	Greig, Dr F. J., Lt.-Col. R.A.M.C., 16 Melville Terrace, Stirling,	1906
	Gunn, Dr J. A., 39 Morningside Drive,	1906
	Guthrie, Dr A. Cowan, 21 Pilrig Street,	1888
190	Hamilton, Dr J. R., Elm House, Hawick,	1879
	Hamp, Dr J. Walton, Penn Road, Wolverhampton,	1886
	Harley, Dr R. J., 102 Preston New Road, Blackburn,	1905
	Harlin, Dr Francis W., Crow's Nest, Near Loowoomba, Queensland,	1900
	Harper, Dr R. A. J., 135 Abbey Road, Barrow-in-Furness,	1901
195	Harvey, Dr Charles, Beckford Lodge, Sav-la-Mar, Jamaica, W.I.,	1889
	Harvey, Dr James, 7 Blenheim Place,	1891
	Havelock, Dr John G., Sunnyside, Montrose,	1888
	Hay, Dr Henry, 11 Great King Street,	1879
	Hellier, Dr J. B., 27 Park Square, Leeds,	1904
200	Helm, Dr John H., Clarence Cottage, Ratho,	1888
	Helm, Dr R. Dundas, 13 Portland Square, Carlisle,	1892
	Helme, Dr G. Edgar, Gloucester House, Rusholme, Manchester,	1895
	Henderson, Dr Alexander, c/o Rev. George Henderson, M.A., B.D., U.F. Manse, Monzie, Crieff,	1891
	Hewat, Dr A. Middleton, Borough Isolation Hospital, Derby,	1907
205	Hewetson, Dr J., Holmfield, Reigate,	1881
	Hindmarsh, Dr Edwin, Mozufferpore, Tirhoot State Railway, Bengal, India,	1895
	Hobson, Dr H. Overton, Villa Sakkara, Helouan, Egypt,	1901
	Hoggan, Dr Robert, Liberton Park, Liberton,	1894
	Howard-Jones, Dr M. H., 17 Cornwall Street,	1910
210	Hughes, Dr Percy T., County Asylum, Bromsgrove, Worcestershire,	1895
	Hunter, Dr A. J. Gordon, St Catherines, Linlithgow,	1910
	Hutchison, Dr Alice M., 204 Bruntsfield Place,	1905
	Inglis, Dr Elsie M., 8 Walker Street,	1901
	Jamieson, Dr Hugh, 1 Strathearn Road,	1889
215	Jamieson, Dr J. Boyd, 43 George Square,	1900
	Jardine, Dr F. E., Evelyn Cottage, Penicuik,	1910
	Jardine, Dr Robert, 20 Royal Crescent, Glasgow, W.,	1897
	Johnston, Dr Robert B., Bishopyards, Penrith,	1903
	Johnston, Dr R. J., 18 George Square,	1899
220	Johnstone, Dr R. W., 10 Alva Street,	1903
	Jones, Dr W. Llewellyn, 58 Thomas St., Merthyr-Tydvil,	1903
	Keay, Dr J. W., 13 Alva Street,	1903
	Keir, Dr Ian C., The Limes, Melksham, Wilts,	1903
	Ker, Dr Claude B., City Hospital, Comiston Road,	1894
225	Kerr, Dr J. M. Munro, 7 Clairmont Gardens, Glasgow,	1894
	Kerr, Dr J. Wishart, 107 Greenhead Street, Glasgow,	1901
	King, Dr J. K., The Glen Springs Sanitorium, Watkins, New York, U.S.A.,	1884
	Kirk, Dr Robert, Rowan Bank, Bathgate,	1887
	Kynoch, Professor Campbell, 8 Airlie Place, Dundee,	1892
230	Lackie, Dr James Lamond, 7 Randolph Crescent,	1889
	Langwell, Dr H. G., 4 Hermitage Place, Leith,	1891
	Lawrence, Dr H. Ruthven, South African Union, 14 Buccleuch Place,	1908
	Ledger, Dr A. G. K., Westerton, Coppull, Lancashire,	1906
	Lee, Dr Herbert E., Coolabah, Belmore Street, Burwood, N.S.W., Australia,	1892

		Date of Admission.
235	Littlejohn, Professor Harvey, 11 Rutland Street,	1890
	Lochhead, Dr James, 10 Grosvenor Street,	1904
	Lockhart, Dr F. A. L., 23 Mackay Street, Montreal, Canada,	1890
	Lorimer, Dr Duncan, 74 Bruntsfield Place,	1906
	Low, Dr R. Cranston, 7 Castle Terrace,	1902
240	Lyell, Dr John, 15 Marshall Place, Perth,	1901
	Lyle, Dr R. P. Ranken, 11 Ellison Place, Newcastle-on-Tyne,	1901
	MacArthur, Dr D. G., Aberfeldy,	1903
	M'Call, Dr John, 25a Abercromby Terrace, Portobello,	1886
	M'Cann, Dr F. J., 14 Wimpole Street, Cavendish Square, London, W.,	1896
245	Macdonald, Dr A. G., Uig, Isle of Skye,	1903
	Macdonald, Dr Angus, 27 Manor Place,	1897
	Macdonald, Dr John, Marathon House, Cupar-Fife,	1902
	Macdonald, Dr W. Fraser, 16 Buckingham Ter., Glasgow, W.,	1884
	Macdonald, Dr W. Fraser, Olive Lodge, Polwarth Terrace,	1910
250	M'Ewan, Dr Peter, 1 Pavilion Terrace, Scarborough,	1905
	M'Gibbon, Dr John, 24 Melville Street,	1902
	MacGlashan, Dr J. Keith B., Maternity Hospital.	1910
	MacGregor, Dr Alastair, Eagle Cliff, Greenhithe, Kent,	1905
	MacGregor, Dr A. V., Durham House, West Hartlepool,	1895
255	Macgregor, Dr G. S., 15 Buckingham Terrace, Glasgow, W.,	1888
	M'Ilwraith, Dr Kennedy C., 30 Prince Arthur Avenue, Toronto, Canada,	1901
	Mackay, Dr W. B., 23 Castlegate, Berwick-on-Tweed,	1899
	M'Kendrick, Dr Archd., 27 Chalmers Street,	1906
	Mackenzie, Dr Robert, Napier, Nairn,	1887
260	Mackenzie, Dr T. C., Ruigh-Ard, Inverness,	1900
	M'Kerron, Dr R. Gordon, 1 Albyn Place, Aberdeen,	1896
	Mackie, Dr George, Boyd's Lodge, Malvern, Worcestershire,	1900
	Mackin, Dr Patrick, 12 Ingestre Street, Wellington, New Zealand,	1895
	Mackness, Dr G. Owen C., Fort Street House, Broughty-Ferry,	1887
265	Maclagan, Dr D. W., Kaponga, Taranaki, New Zealand,	1901
	M'Laren, Dr John, 39 Lauriston Place.	1910
	M'Larty, Dr Malcolm, 7 Bellevue Place,	1900
	M'Lean, Dr Archibald, Crosshouse, Kilmarnock,	1890
	Maclean, Dr Ewen J., 12 Park Place, Cardiff,	1902
270	Macleod, Dr J. A., The Asylum, Inverness,	1907
	M'Master, Dr A. C., Erinholm, Hopetoun St., Auckland, New Zealand,	1906
	Macmillan, Dr John, 48 George Square,	1897
	M'Morland, Dr John B., 19 Merchiston Gardens,	1906
	M'Morland, Dr R. P., 19 Merchiston Gardens,	1907
275	MacRae, Dr John, Lynwood, Murrayfield,	1893
	MacVie, Dr S., Herbertknowe, Chirnside,	1881
	M'Watt, Dr John, Morelands, Duns,	1879
	Macwatt, Dr R. C., Lt.-Colonel I.M.S., c/o Messrs Henry S. King & Co., 9 Pall Mall, London, S.W.,	1885
	Malcolmson, Dr Alexander M., Dalveen, Corstorphine,	1901
280	Mallace, Dr A. C., 30 Morningside Park,	1908
	Marshall, Dr G. Balfour, 19 Sandyford Place, Glasgow,	1891
	Martin, Dr Angus, 25 Northumberland Square, North Shields,	1906
	Martin, Dr Charles, Dagenham House, Newton Abbot, South Devon,	1892
	Martin, Dr J. W., Charterhall, Newbridge, Dumfries,	1887
285	Matheson, Dr A. A., 41 George Square,	1887
	Matheson, Dr R. Murdoch, 33 Buccleuch Place,	1897

		Date of Admission.
	Mathewson, Dr G. D., 6 Castle Terrace,	1905
	Meikle, Dr J. Hally, 12 Midmar Gardens,	1906
	Menzies, Dr David, 20 Rutland Square,	1877
290	Menzies, Dr W. Menzies, 20 Rutland Square,	1910
	Michael, Dr Gustave, Bayham House, 135 West End Lane, West Hampstead, London, N.W.,	1885
	Millard, Dr W. W., Middlefield House, Leith Walk,	1884
	Miller, Dr Alexander, 1 Royal Terrace, Crosshill, Glasgow,	1902
	Miller, Dr W. H., 51 Northumberland Street,	1886
295	Milligan, Dr Harley P., Hessle, East Yorks,	1908
	Milne, Dr W. M., 10 Newington Road,	1898
	Mitchell, Dr C. R. P., The Chase, Malvern,	1906
	Mitchell, Dr G. B., 1 Skinner Street, Whitby,	1893
	Montgomery, Dr John, The Highlands, Balsall Heath, Birmingham,	1895
300	Moorhouse, Dr J. Ernest, 6 Melville Terrace, Stirling,	1892
	More, Dr John, Rothwell, Kettering,	1908
	Morison, Dr Albert E., Mowbray Villa, Sunderland,	1888
	Morris, Dr S. Glanville, Bryn-y-Delyn, Mardy, Glamorganshire,	1894
	Moses, Dr O. St John, Medical College, Calcutta, India,	1900
305	Mowat, Dr John, 5 Hope Park Terrace,	1884
	Mowat, Dr R. S., 40 Raeburn Place,	1910
	Munro, Dr J. Ramsay, Sutterton, Boston,	1901
	Murray, Dr James, 51 Great King Street,	1879
	Murray, Dr W. P., 12 Merchiston Crescent,	1910
310	Napier, Dr A. D. Leith, 28 Angas Street, Adelaide, South Australia,	1878
	Nicholson, Dr H. Oliphant, 20 Manor Place,	1900
	Ogden, Dr O. Watson, 38 Jesmond Road, Newcastle-on-Tyne,	1900
	Ogilvy, Dr Stewart Grant, Fairmont, Fauldhouse,	1895
	Oliphant, Dr E. H. Lawrence, 23 Newton Place, Glasgow,	1885
315	Orbell, Dr Ronald S., 12 Reed Street, Oamaru, New Zealand,	1907
	Orr, Dr John, Heather Lea, Clarendon Road, Eccles, Lancs.,	1887
	Orr, Dr W. Basil, 13 Braid Road,	1889
	Paterson, Dr G. Keppie, 19 Albany Street,	1882
	Paterson, Dr G. W. Simla, 147 Bruntsfield Place,	1896
320	Patterson, Dr F. Porter, F.R.C.S.Ed., 203 Dominion Trust Building, Vancouver, B.C.,	1909
	Pearson, Dr C. Mowbray, 14 Manor Place,	1902
	Peddie, Dr H. Anderson, 24 Palmerston Place,	1882
	Perigal, Dr Arthur, New Barnet, Herts,	1887
	Pirie, Dr John, Moss Side, New Deer, Aberdeenshire,	1888
325	Playfair, Dr John, 5 Melville Crescent,	1873
	Poole, Dr T. D., North Side House, Linthwaite, Huddersfield.	1890
	Porritt, Dr E. E., Wanganui, New Zealand,	1897
	Porteous, Dr J. Lindsay, 83 Warburton Avenue, Yonkers, New York,	1875
	Porter, Dr Frederick, 65 Morningside Road,	1904
330	Prentice, Dr W. H., Brunswick Terrace, Broad Street, Pendleton, Manchester,	1901
	Price, Dr A. W. Gordon, 9 Grange Road,	1890
	Price, Dr E. F. T., 1 Middleby Street,	1890
	Primrose, Dr Alex., 100 College Street, Toronto, Canada,	1887
	Pringle, Dr J. Hogarth, 172 Bath Street, Glasgow,	1886
335	Proudfoot, Dr Thomas, 30 Lauriston Place,	1884
	Rabagliati, Dr A. H., c/o H. A. Jeffrey, Esq., Solicitor, 13 Cheapside, Bradford, Yorkshire,	1903

		Date of Admission.
	Reid, Dr H. S., 6 Kew Terrace,	1907
	Reid, Dr W. L., 7 Royal Crescent, W., Glasgow,	1880
	Rendell, Dr Herbert R., P.O. Box 606, St John's, Newfoundland,	1882
340	Ritchie, Dr James, 22 Charlotte Square,	1880
	Ritchie, Dr W. T., 9 Atholl Place,	1899
	Robarts, Dr Henry H., 1 Wemyss Place, Haddington,	1904
	Roberton, Dr Ernest, Cotele House, Symond Street, Auckland, New Zealand,	1887
	Roberts, Dr D. Lloyd, 11 St John Street, Manchester,	1906
345	Roberts, Dr Ernest T., Glencloy, Brownside Road, Cambuslang, Glasgow,	1890
	Roberts, Dr R. W., Bronwylfa, Grange Street, Port Talbot, Glamorganshire,	1894
	Robertson, Dr George, Brachead, Viewfield Place, Dunfermline,	1901
	Robertson, Dr Robert, 26 Royal Circus,	1897
	Robertson, Dr W. B., St Anne's, 101 Thurlow Park Road, Dulwich, London, S.E.,	1870
350	Robertson, Dr W. G. Aitchison, 2 Mayfield Gardens,	1889
	Robinson, Dr H. Shapter, Talfourd House, 78 Peckham Road, Camberwell, London, S.E.,	1890
	Ronaldson, Dr James Bruce, Charing Cross Hospital, London,	1911
	Ronaldson, Dr T. R., 8 Charlotte Square,	1877
	Rosa, Dr Albert F., 28 Pitt Street,	1893
355	Ross, Dr A. Aitken, 8 Newington Road,	1910
	Ross, Dr T. W. E., 65 Morningside Drive,	1904
	Russell, Dr W., 3 Walker Street,	1890
	Saleeby, Dr C. W., 13 Greville Place, St John's Wood, London, N.W.,	1902
	Sandstein, Dr Alfred C., 23 Latimer Square, Christchurch, New Zealand,	1898
360	Saunders, Dr F. A., Hill Street, Grahamstown, Cape Colony, South Africa,	1885
	Sclater, Dr W. O., Palmer Memorial Hospital, Jarrow-on-Tyne	1906
	Scott, Dr Alexander, The Firs, Broxburn,	1906
	Scott, Dr T. R., 13 High Street, Musselburgh,	1880
	Secord, Dr E. R., 112 Market St., Brantford, Ontario, Canada,	1902
365	Shannon, Dr David, 270 Bath Street, Glasgow,	1910
	Shaw, Dr C. J., Argyle and Bute Asylum, Lochgilphead,	1903
	Shearer, Dr Alfred, The Bank, Newtown, N. Wales,	1898
	Shepherd, Dr F. W., Sydenham House, Pontefract Lane, Leeds,	1908
	Simpson, Dr Archibald, 16 Swan Hill, Shrewsbury,	1906
370	Simpson, Dr F. D., 9 Hampton Terrace,	1900
	Slight, Dr J. D., 163 London Road, Leicester,	1898
	Sloan, Dr Allen T., 22 Abercromby Place,	1893
	Sloan, Dr S., 5 Somerset Pl., Sanchiehall St., West, Glasgow,	1887
	Smart, Dr David, 74 Hartington Rd., Sefton Park, Liverpool,	1882
375	Smith, Dr G. H. Walton, Pendower, Oxford St., Paddington, Sydney, Australia,	1892
	Smith, Dr Gains T., 71 Church Street, Moncton, New Brunswick, Canada,	1891
	Smith, Dr James, 4 Brunton Place,	1891
	Smith, Dr John, Kirk Wynd, Kirkcaldy,	1885
	Smith, Dr W. Ramsay, Central Board of Health, Adelaide, South Australia,	1892
380	Smith, Dr W. Torrance, 70 Woodlands Road, Aigburth, Liverpool,	1906

		Date of Admission.
	Sneddon, Dr William, Logan Bank, Cupar-Fife,	1888
	Somerville, Dr C. W., Glencairn, Dalkeith,	1902
	Somerville, Dr James W., The Grange, Galashiels,	1902
	Stark, Dr J. Nigel, 4 Newton Place, Charing Cross, Glasgow,	1908
385	Stephen, Dr W. A., Loftus-in-Cleveland, Yorkshire,	1894
	Stephenson, Professor Wm., 3 Rubislaw Terrace, Aberdeen,	1861
	Stevens, Dr John, 78 Polwarth Terrace,	1894
	Stewart, Dr A. D., 8 Brougham Place,	1906
	Stewart, Dr J. S., 15 Merchiston Place,	1884
390	Stewart, Dr R., 25 George Square,	1885
	Stirling, Dr R., 4 Atholl Place, Perth,	1892
	Strain, Dr Arthur C., Lathland House, West Hartlepool,	1904
	Stumbles, Dr H. M., Amble House, Amble, Northumberland,	1903
	Sturrock, Dr J. F., Arima, Broughty-Ferry,	1887
395	Sullivan, Dr John, 34 Gilmore Place,	1902
	Tannahill, Dr W. B., 40 Margaret Street, Greenock,	1907
	Taylor, Dr David R., St Helen's, Ayton,	1896
	Taylor, Dr William, 12 Melville Street,	1868
	Taylor, Dr W. Macrae, 8 Melville Street,	1895
400	Teacher, Dr Charles C., Craigend, North Berwick,	1887
	Temple, Dr G. H., Ailanthus, Weston-super-Mare,	1888
	Tennant, Dr John, 89 Frodingham Road, Scunthorpe, Doncaster,	1902
	Thatcher, Dr C. H., 8 Melville Crescent,	1877
	Thatcher, Dr L. H. F., 8 Melville Crescent,	1910
405	Thin, Dr Robert, 25 Abercromby Place,	1891
	Thomas, Dr G. Crewdson, 10 Chester Square, London, S.W.,	1899
	Thompson, Dr F. E., 20 Park Avenue, Montreal, Canada,	1902
	Thompson, Dr Herbert P., 7 Springfield Avenue, Harrogate,	1906
	Thompson, Dr James L., Castlemaine, Victoria, Australia,	1894
410	Thomson, Dr A. D. R., 19 Bridge Street, Musselburgh,	1877
	Thomson, Dr John, 14 Coates Crescent,	1887
	Thomson, Dr T. J., 3 Greenhill Park,	1902
	Thyne, Dr T. J., 16 Randolph Crescent,	1887
	Tod, Dr John, 67 Ferry Road, Leith,	1892
415	Tristan, Dr R. J., 28 Carolgate, Retford, Notts,	1887
	Tuke, Dr A. L. S., 12 Comely Park, Dunfermline,	1911
	Turnbull, Dr A. E., Clearmount, Broadway, Dorset,	1909
	Underhill, Dr T. Edgar, Dunedin, Barnt Green, Worcestershire,	1879
	Vassie, Dr Alexander H., 98 Priory Road, West Hampstead, London, N.W.,	1891
420	Venters, Dr Isabel, 6 Archibald Place,	1907
	Vickery, Dr W. H., 1 Trewartha Park, Weston-super-Mare,	1892
	Walker, Dr Arthur S., School Board Offices, Leith,	1906
	Wallace, Dr Abraham, 39 Harley Street, London, W.,	1879
	Wallace, Dr R. W. L., The Hospital, Assouan Reservoir, Upper Egypt,	1905
425	Watson, Dr B. P., 7 Castle Terrace,	1902
	Watson, Dr R. H., Trereiffe, Roche, R.S.O., Cornwall,	1896
	Waugh, Dr John, 36 Finsbury Pavement, London, E.C.,	1881
	Webster, Dr A. D., 18 Minto Street,	1881
	Webster, Prof. J. C., 706 Reliance Building, 100 State Street, Chicago, U.S.A.,	1889
430	Wemyss, Dr H. L. Watson, Westbourne House, Broughty Ferry,	1910
	White, Dr A. L., Tantallon, Manchester Road, Castleton, near Manchester,	1903
	Wilkie, Dr James, Selville House, Portobello,	1897
	Wilkinson, Dr George, 3 Dingle Hill, Liverpool, S.,	1889

		Date of Admission.
	Will, Dr J. C. Ogilvie, 17 Bon-Accord Square, Aberdeen, .	1867
435	Williams, Dr J. T., Llwynypia, Rhondda Valley, Glamorganshire, ...	1904
	Willcox, Dr F. Mayes, 8 Strathearn Road, . . .	1902
	Wilson, Dr James, 53 Inverleith Row, ...	1889
	Wise, Dr Robert, 37 Vartry Road, Stamford Hill, London, N.,	1890
	Wood, Dr Thomas, 182 Ferry Road, ...	1886
440	Woodside, Dr J. T., 13 Meadow Street, Aberkenfig, R.S.O., Glamorganshire, ...	1898
	Yoe, Dr Richard T., 415 W. Chestnut Street, Louisville, Kentucky, U.S.A., ...	1891
	Young, Dr H. C. Taylor, 221 Macquarrie Street, Sydney, New South Wales, ...	1900
443	Young, Dr James, 4 Torphichen Street, . . .	1907

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TRANSACTIONS

OF THE

EDINBURGH OBSTETRICAL SOCIETY,

FOR SESSION LXXII., 1910–1911.

Meeting I.—November 5, 1910.

Dr Haultain, *President, in the Chair.*

I. *The Treasurer* (*Dr Wm. Craig*) made his Annual Statement, which is given below:—

INCOME.

Balance from Session 1908-1909,					£541	3 4
Arrears,	£8	5	0			
Bank Interest on Deposit Receipts,	2	1	2			
Interest on Consols,	5	19	8			
Interest on N.B.R. Debenture Stock,	8	9	6			
Entrance Fees from 12 new Ordinary Fellows,	12	12	0			
Annual Contributions from 369 Ordinary Fellows,	92	5	0			
Composition for Life-Membership from 1 Ordinary Fellow,	5	5	0			
Transactions sold,	5	7	4			
					140	4 8
					£681	8 0

EXPENDITURE.

Corporation Duty,	£0	16	9			
Income Tax,	0	4	8			
Shorthand Reporter,	5	5	0			
Commission to Collector,	1	8	0			
Dr B. P. Watson, for Illustrations,	1	17	6			
Door-keeper's Salary,	1	12	0			
Oliver & Boyd's Account for Vol. XXXV. of Society's *Transactions* (560 copies),	85	19	0			
Oliver & Boyd's Account for Printing Billets, Postages, etc.,	24	2	6			
Rent of Rooms,	4	10	0			
Waterson & Sons' Account,	2	5	3			
A. H. Baird's Account,	1	4	0			
A. Frazer's Account,	0	16	0			
A. Locke & Son's Account,	0	3	6			
Editor—Postages,	0	5	0			
					£130	9 2
*Balance to New Account,					550	18 10
					£681	8 0

* Of this Balance, the sum of £250, 10s. 6d. is invested in 2½ per cent. Consols, and the sum of £254, 16s. in 3 per cent. N.B.R. Debenture Stock.

The accounts were audited by *Drs James Ritchie* and *Barbour*, and found correct.

The Treasurer then read his report, which is as follows:—

TREASURER'S REPORT FOR SESSION 1909-1910.

The Society commenced the past Session with a balance in its favour of . . .	£541	3	4
The total Income for the Session has been .	140	4	8
The total Expenditure for the Session has been .	130	9	2
It will thus be seen that the Income has exceeded the Expenditure by	9	15	10
which sum, added to the balance from the preceding Session, leaves a balance of .	550	18	10
with which to begin the present Session.			

As this is the last Report which I shall have the honour to present to the Society, after having held the office of Treasurer for thirty-five years, I hope it is not out of place to contrast briefly the state of the finances of the Society at present with what they were thirty-five years ago, when I had the honour of being elected your Treasurer.

The balance in favour of the Society thirty-five years ago was	£20	12	11
It is now	550	18	10
An increase of	530	5	11

This, however, by no means represents the whole of the increase, for it must be borne in mind that, for thirty-three years, the Fellows of the Society have received copies of the *Transactions* delivered free at their houses, and for the last thirty of these years, the volume has been beautifully bound. This has been accomplished without increasing either the entrance fee or the annual subscription. The reason why the Society has been able to accomplish all this is the fact that

each Ordinary Fellow receives a copy of the *Transactions* FREE, irrespective of the quarter of the Globe in which he may reside.

The history of the publication of the *Transactions* may interest the younger Fellows of the Society.

Volume I., which contains the *Transactions* for Session 1868-69, was published in 1870 at 7s. 6d. per volume, but only 5s. was charged to the Fellows of the Society. The Society lost fully £40 by this transaction.

Volume II., which contains the *Transactions* for Sessions 1869-70 and 1870-71, was published in 1872, at 15s. per volume, but only 10s. 6d. to the Fellows of the Society. By this transaction the Society lost fully £40, and at the end of the Session 1872-73 was in debt to the Bank £17, 16s.

Volume III., which contains the *Transactions* for Sessions 1871-72, 1872-73, and 1873-75, was published in 1875 by Messrs Maclachlan & Stewart entirely at their own risk (for the Society had no funds for such a purpose). The price was 15s., but only 10s. 6d. to the Fellows of the Society. By this transaction Messrs Maclachlan & Stewart lost fully £30, and positively refused to publish any more volumes. As the Society had no funds with which to carry on the publication of the *Transactions*, a motion was made to increase the annual subscription for this purpose, but fortunately this motion was rejected.

Volume IV., which contains the *Transactions* for Sessions 1874-75, 1875-76, and 1876-77, was published in 1878. The Society undertook the whole expense of the publication of this volume, after receiving subscriptions of half-a-guinea from about 40 Fellows. Only about 80 copies were printed. This volume cost the Society £83, 6s., but with subscriptions and sales, £64, 18s. 6d. was realised, so that the total loss was only £18, 7s. 6d., or £2, 5s. 5d. less than what the Society possessed in November 1875.

The *Transactions* for Session 1877-78 and the two following

Sessions were sent annually free to all the Fellows, but only in paper cover. This experiment of sending the *Transactions* free to all the Fellows proved so successful financially, that at the end of three years the Society resolved to bind the volumes; and this has been done for the past thirty years, during which time the Funds of the Society have increased from £62, 2s. 9½d. to £550, 18s. 10d., an increase of £488, 16s. 0½d.

During these thirty-five years the number of Ordinary Fellows has risen from 109 to 443, an increase of 334 Ordinary Fellows.

There can be no doubt that the increase in membership and finances has been mainly due to the *free* distribution of the *Transactions* to the Fellows of the Society.

At November 1875 the amount possessed by the Society was £20, 12s. 11d.; five years later,

November 1880, it was . .	£62	2	9½
November 1885, it was . .	85	9	8
November 1890, it was . .	184	10	6
November 1895, it was . .	319	9	0
November 1900, it was . .	452	3	10
November 1905, it was . .	563	6	2
November 1910, it is . . .	550	18	10

I trust these facts will prove a stimulus to the Fellows of the Society to make the *Transactions* more and more valuable, so that during the years to come the prosperity of the Society may be even greater than in the past.

Dr Haultain proposed, and *Sir Alexander Simpson* seconded, a hearty vote of thanks to Dr Craig for his invaluable services as Treasurer during the last thirty-five years. The President wished to convey to him the cordial congratulations of the Society on his Thirty-fifth Annual Report, and their regret that he was no longer to be their Treasurer.

Dr Craig returned his warm thanks.

II. The Society then proceeded to the election of Office-bearers for the present Session, and the President announced the result as follows:—*President*, Dr Haultain; *Vice-Presidents*, (*Senior*) Dr Lamond Lackie, (*Junior*) Dr Brewis; *Treasurer*, Dr G. Barbour Simpson; *Secretaries*, Dr Angus Macdonald, Dr B. P. Watson; *Librarian*, Dr Lamond Lackie; *Editor of Transactions*, Dr Oliphant Nicholson; *Members of Council*, Professor Stephenson, Dr Frederick Porter, Dr F. D. Simpson, Dr Haig Ferguson, Dr Wm. Craig, Dr Lawrence Oliphant, Professor Kynoch, Dr Kenmure Melville.

III. The following gentlemen were elected Ordinary Fellows of the Society:—J. Keith B. MacGlashan, M.B., Ch.B., Maternity Hospital, Edinburgh; Matthew R. Drennan, M.A., M.B., Ch.B. The Residency, Royal Infirmary, Edinburgh; Frank E. Jardine M.B., Ch.B., Evelyn Cottage, Penicuik; R. S. Mowat, M.D., 46 Raeburn Place, Edinburgh; R. C. Alexander, M.B. Ch.B., 15 Hartington Gardens.

IV. Presidential Address.

A RETROSPECT AND COMPARISON OF THE PROGRESS OF MIDWIFERY AND GYNÆCOLOGY.

By F. W. N. Haultain, M.D., F.R.C.P.

To occupy the chair of this renowned Society, and to be classified with the many eminent men who have adorned it, is indeed an honour of which I am truly proud. Honours, doubtless, are gratifying, but, as a rule, carry in their train responsibilities corresponding to their magnitude. Alas! the position I am in this evening is no exception.

According to custom, it is my duty to deliver an address, a privilege I feel far from able to cope with in a manner commensurate with the occasion. As necessity compels, however,

and it has been my fortunate lot to commence the study of medicine in the late seventies, which may be said to be the date of the birth of modern gynæcology, I have taken the opportunity thus offered of comparing the work of our Society before and after that period, which practically divides its existence into two equal halves.

Before 1878 our Society was mainly concerned (as the name implies) in obstetrics. Gynæcology might be designated in Gilbertian phraseology to be "a thing of plugs and douches." Since that date it has gradually developed, till now it bulks most largely in our deliberations. It has risen from a mere appendage to medical practice to form one of the most prominent specialities of medical science, and has passed from the realms of medicine in its true sense entirely into that of surgery.

In this striking metamorphosis we may proudly assert, without fear of contradiction, Edinburgh has played by far the most prominent part. Without J. Y. Simpson, Lister, Keith, and Tait, it is impossible to think of modern gynæcology. The plug, douche, and pessary would still reign supreme. Fibroids and ovarian tumours would still claim their victims with ruthless tyranny, while we as general practitioners would still have added to our sorrows the irksome duty of, with folded hands, trying, with kindly words, to mitigate their present suffering with the problematical hope of a more comfortable old age. Simpson's anæsthesia and Lister's antiseptics made it possible for Thomas Keith, by his wonderful conscientious assiduity, to make, in the face of much opposition and popular prejudice, ovariotomy one of the most successful operations in modern surgery, and at the same time to lay the foundations of the modern method of performing hysterectomy, which may fairly lay claim to be at the present time the greatest surgical boon to suffering womankind.

No one who was privileged to see his one and only semi-

public ovariotomy will forget it. The big theatre of the present Infirmary filled to discomfort; its well festooned with the entire staff, which included many brilliant surgeons; the pale, gaunt surgeon with anxiety stamped on every feature; his weird movements as he manipulated the intestines in hot-soaked towels; the entire stillness, broken only by the hissing of the carbolic spray or the fizzing of the seared pedicle—all seemed to savour more of a wizard's séance than a reality. Alas! the result was unsuccessful, and never again was the opportunity offered of seeing the great surgeon at work.

By the same means Lawson Tait, with his brilliant genius, practically proved the ready accessibility of the peritoneum to surgical interference.

With his intrepid fearlessness he penetrated this sanctuary with scant courtesy in an almost ruthless manner. With his marvellous dexterity, through a 2-inch incision, he enucleated inflammatory appendages bound down by dense adhesions, and showed that contamination of the peritoneum by pus was not necessarily fatal. By his brilliant success he did much to develop general abdominal surgery, particularly in regard to inflammatory conditions.

To Keith and Tait modern gynæcology may be said to owe its high position. To the former no one would be readier to assign the premier position than the latter. From his own lips I have heard his eulogy, so characteristic of the bigness of the man. To have known and watched both is indeed a privilege. Two more different natures could not well be imagined. Keith careful, anxious, retiring almost to a fault; Tait intrepid, confident, and aggressive. Both, however, striving at the same ideal with equal success. Surely an object-lesson of priceless value!

As has already been said, since the late seventies gynæcology has been completely transformed, and has emerged from a medical to a surgical science, and it has been interesting to have watched its development through the phases of antisepsis

and asepsis, the former being most aptly illustrated by the carbolic spray, the latter by rubber gloves. This may at once be said to have been the Alpha and Omega of the metamorphosis.

Up till this time (as the records of our Society show) surgical procedure was very meagre, yet there was evident throughout a latent surgical vitality ready to burst into full vigour whenever opportunity offered. Nothing could be more convincing of this than the attitude of Sir J. Y. Simpson, who in July 1863 said as follows:—

"In the discussion which had recently taken place in the Med. Chirurgical Society some of their brethren seemed aggrieved because he had spoken of the opposition that surgeons here had so long offered to the introduction of the operation. Yet, most assuredly, they had so spoken and written against ovariotomy and its performance that it had been too long kept back out of the list of legitimate operations.

"He had also written and lectured in favour of it. It might be one in three or four of the cases will always die, but one could not doubt that the operation will become more perfect and less fatal as surgeons go on applying themselves with improvement.

"He also strongly advocated removal of the cyst in preference to tapping."

At the same meeting Dr Keiller stated he had asked Professors Syme and Spence, then Infirmary surgeons, to remove ovarian tumours, but they absolutely refused.

In 8th June 1840 the note of the first operation specimen brought before the Society was a case of cauliflower excrescence of the os uteri, successfully removed by Dr Lewins, and at the same meeting Dr J. Y. Simpson showed a fibrous polypus, the size of a nut, he had cut away.

The next note of an operation was the more ambitious

procedure of Cæsarean section by Dr Ross, Invergordon, on 8th March 1842. The child lived, but unfortunately the mother died on the third day "from imprudent exposure"

In November 1843 Dr J. Y. Simpson brought forward statistics of 12 cases of laparotomy performed in Britain, with 4 deaths.

In January 1856 a letter from Dr Clay was read, giving the result of 69 collected cases of ovariotomy.

In 1862 a specimen of hydatid of the omentum was shown, successfully removed by Dr West, Birmingham, which was thought before operation to be an ovarian cyst.

The first example of vaginal surgery was communicated by Dr Alexander Simpson, in which Dr J. Y. Simpson opened a supposed pelvic hæmatoma through the vaginal vault and found a gestation sac, which he successfully removed. He at the same time related a similar case of extra-uterine pregnancy which J. Y. Simpson attempted to remove through the abdominal wall by means of caustic, but the woman sank. In this instance the woman had a history of injury during the early months of gestation, and it was thought the uterus had been fissured.

A note of a successful Cæsarean section by Dr Anderson, Jamaica, was read in December 1863, and notes of 2 cases of ovariotomy in 1867 and 1 in 1876 were also intimated.

It is also interesting to see that Dr Alexander Simpson at this time (1876) had performed 3 ovariotomies, with 1 death.

The above is more or less a complete record of the surgical performances brought before the Society.

From this period the *Transactions* of the Society gradually show increasing examples of surgical procedure, till they literally teem in the later volumes. Thus, in 1908-9, 41 cases of abdominal section alone are recorded.

The development of surgical technique, as shown in our

Transactions, is extremely interesting. Thus in ovariotomy, which may be looked upon as the doyen of abdominal surgery, we have a kaleidoscopic picture.

The pedicle was first secured extra-abdominally by clamps, then it was clamped and seared, and dropped into the abdominal cavity; later, to this was added, by Angus Macdonald (one of our most brilliant operators), the ligation of the vessels by silver wire and silk. The cautery was then discarded, and the pedicle secured by silk, catgut, or linen, tied *en bloc* by a variety of knots, such as the Staffordshire and Bantock, and then dropped into the peritoneal cavity. From this we have advanced to ligation in parts and careful inversion of the raw stump, with subsequent covering by peritoneum.

The abdominal wound in the same way was shown to be diversely closed by pins, silver wire, silk, and silkworm gut *en masse*; now the preponderance of opinion is more sentimental, and tends towards catgut in layers, with a final subcutaneous continuous suture.

Perhaps in no instance has change in procedure been so marked as in the after-treatment. At first it was so rigidly severe as to resemble more the tortures of a Chinese prison than the philanthropic endeavours of a charitable profession.

For a fortnight the patient was kept rigidly on her back; the first forty-eight hours after the operation not even a drop of water was allowed to pass over the lips, in spite of the intense thirst always complained of; while the bowels were locked for eight days. How different from the present system! where free movement is permitted from the time the patient is returned to bed, fluids given whenever they can be retained, and the bowels freely opened on the second or third day.

During the eighties and early nineties drainage by the abdomen or vagina was advised in even the simplest cases; now it is practically universally discarded. Many other similar

differences might be recorded, but sufficient has been said to show the marked development which has taken place within a comparatively short period.

As is natural to expect, the sacred precincts of the peritoneum having been safely entered, they were rapidly more freely explored. Inflamed and adherent tubes and ovaries were enucleated and removed, and extra-uterine gestation recognised and extirpated. For a time uterine appendages were removed in a manner similar to the appendix in later years, in a wholesale way, without valid reason in many cases. This caused a reaction and swing of the pendulum to the opposite conservative extreme, from which it is doubtful if it has yet returned to the normal mean.

In the early eighties attention began to be turned to the removal of uterine fibromyomata. It is interesting to note in this connection a paper by Dr A. R. Simpson on the treatment of these tumours, read on 14th November 1877, in which no mention of removal by the abdomen is made. Thomas Keith, however, in his quiet, unostentatious way, now commenced to lay the foundation of a steady attack on these curses to womankind, which was so ably sustained by Angus Macdonald and A. R. Simpson among our Fellows.

By stripping the peritoneum from the stump before clamping it extra-abdominally, he had acquired a success unapproached by any other living operator (a less than 10 per cent. mortality). Poor health and the excessive strain told on his nerve, however, before he perfected the operation which he absolutely discarded, and degenerated to the treatment of fibromyomata by Apostoli's electrical methods.

Gradually, as in ovariotomy, the extra-abdominal treatment of the stump gave way to the intra-peritoneal, with its varying but progressive refinements, till the operation as practised by us now has as nearly reached perfection as one can possibly conceive. If gynæcological surgery had done nothing else than

reach the present stage of the treatment of fibroids, anyone might be proud to be identified with it.

In June 1882 Professor A. R. Simpson described the first case before the Society, of Vaginal Hysterectomy for Cancer, which, however, proved fatal. In 1884 Angus Macdonald described the first successful case by this route. It was not, however, till well on in the nineties that this suddenly became the fashionable route for abdominal pelvic surgery. For a short time it became a furore; but as an alternative to the abdominal route, except in very special cases, it has ceased to command favour with most surgeons.

Sufficient has been said by even touching thus lightly on the advances of gynæcology within the last thirty years to convince anyone of its extraordinary strides, as reflected in the *Transactions* of our Society, and one may now turn shortly to a review of its sister—I might rather say mother—subject, midwifery.

Here one may at the outset say a very different state of matters exists. Little development has taken place. This statement, however, cannot be made in the spirit of reproach. Comparison is impossible. In gynæcology we have to deal with a science from its birth, almost conception, through a lusty childhood and vigorous youth. In midwifery, on the other hand, we have a subject in the robust and mature manhood of thousands of years. We may congratulate ourselves, therefore, if we can say that it still even shows signs of development and progress in its advanced years. So far as the Society's *Transactions* show, there was little left for us to teach our predecessors, as embodied in those ten worthies who on 4th December 1839 met together in the New Town Dispensary, Thistle Street, "to consider the propriety of establishing an Obstetrical Society in Edinburgh."

In a general way we might be disposed to flatter ourselves that the management of pregnancy, labour, and the puerperium

has advanced since the late seventies. Antiseptics have made a great revolution in our methods. The axis-traction rods to the forceps have added much to the utility and safety of those instruments. The use of hot water has greatly shorn post-partum hæmorrhage of its terrors; and the rapid advance of surgical procedure, by abdominal and other methods, has rendered possible the safe delivery of cases otherwise apparently hopeless. All of these might be legitimately quoted as improved developments of the last thirty years, and naturally one would expect to find it reflected in the statistical tables of the Registrar-General.

When these are consulted, however, one is staggered at the figures. As the table shows (Table I., p. 27), the mortality from puerperal causes in Scotland is as high as it was fifty years ago, viz. about 5 per thousand, or 1 in 200 cases.

If one groups the figures before and after 1878 (which is the arbitrary division of this address), we find the general puerperal death-rate in Scotland 4·8 per thousand, and 4·9 since, while from puerperal fever 1·7 per thousand before, and 2·0 since.

Those of Edinburgh are very similar, being from all causes 5·6 per thousand before, and 4·9 since; from puerperal fever 2·1 before, and 1·9 since.

Such figures are most unpleasant reading, and one would like to assume that they may be explained by the unreliability of statistics. Still the figures are there, and explanation difficult. It might be suggested that the registration is now more strict and the grouping of diseases more accurate. This might give a grain of comfort as regards puerperal fever, but not in cases in general, occurring during childbed.

So far as puerperal fever is concerned, the death-rate in England and Wales (see Table II., pp. 28 and 29) is very similar, viz., in 1855, 1·6 per thousand; in 1908, 1·4; and here it must be remembered the great bulk of cases are attended by midwives. The statistics from other countries show a variable

result, but in every instance cited there is a marked diminution, till in Sweden, Holland, and Italy it is under 1 per thousand.

Facts such as these would almost seem to prove that the fever death-rate, in spite of antiseptics, still remains the same in the United Kingdom. It is most unwholesome food for reflection, and requires most careful digestion.

The effects of antiseptics have undoubtedly been proved to be of the greatest benefit in hospital treatment of maternity cases, but in private practice, so far as statistics show, there is little evidence of their being of value. This is easy of explanation, as it is well known that hospitals were hotbeds of contagious infection, a condition the cure of which specially lends itself to the principles of antiseptic treatment. Yet (with very occasional exceptions), in spite of the most rigid antiseptic precautions which only hospitals can command, there is a death-rate from fever of 1 per thousand, as gleaned from the aggregate statistics of five of the most prominent maternity hospitals in the United Kingdom, which show that in 18,820 cases, 18 septic deaths occurred.

In general practice strict antisepsis and asepsis as in hospitals, is in the majority of cases impossible. That a mortality of 2 per thousand exists in practice generally may not be considered large. In fact, when we think of the nature of the process of childbirth, the extensive raw surfaces, and the area in which they occur, it is, in the face of our knowledge of the modes of infection, almost incredible that it is so low.

There can be no doubt that Nature has conferred a special immunity on these parts, by the germicidal properties of the vaginal secretions, and other means we are yet ignorant of, as proved by the extreme rarity of virulent germs in the genital canal, though there are myriads at its orifice. If it were not so, no placental site could escape, not to say perineal and other tears. If the secretions, etc., are normal, it would appear as if severe infection were impossible. On the other hand, should

they have lost their germicidal qualities, as shown by the detection of virulent organisms in the vagina before labour, infection is prone to occur, whether introduced by the attendant or not.

That all infection is preventable is, without doubt, a most desirable working idea to aspire to, but, I am afraid, is Utopian in reality.

No one is more rigidly in favour of the antiseptic management of labour than I. Without it our consciences would make us miserable. To say that the antiseptic management of labour is of no value, in the light of our knowledge of infection, would be absurd, and exceedingly wrong so far as teaching is concerned. Yet there can be no gainsaying the fact that its apparent results fall far below legitimate expectations.

There can be no reasonable doubt that labours in general, in the light of our knowledge of infection and resulting teaching, are more carefully and cleanly treated than formerly when the elements of infection were unknown; and hence it is impossible that direct heterogeneous infection can be so common. Further, the cure of many infected cases by antiseptics must be accepted. In the face of this we can only account for the maintenance of the death-rate at the same percentage by an increase of germs in our surroundings, the result of centralisation in cities and overcrowding.

This is only in keeping with the marked increase of appendicitis, septic pneumonia, and other like affections.

By antiseptics, it seems we have been able to keep the death-rate where it was; without them, the results would be tragic to contemplate.

This is aptly illustrated by the statistics of the mortality from puerperal septicæmia in England and Wales :—

"Up till 1900 there was an increasing number of deaths from sepsis, since then it has decidedly dropped. To the adoption of the Midwives Act this may fairly be said to be

due, the majority of mothers being now attended by women educated in the principles of cleanliness and antisepsis.

"In 1902, when this Act came into force, the death-rate from puerperal sepsis per million women was 118.

"In 1907 it fell to 81. As the 1901 census shows the number of women in England and Wales to be 16,800,000, the fall of 37 per million means that, without estimating actual increase in the number of women, 621 women were saved from this disease alone who would have died in 1902." (Champneys.)

Though 2 per thousand seems but a small percentage of mortality, it shows a terrible toll when the aggregate of birth in this country is considered. To say that it is apparently an irreducible minimum would be unworthy of our profession, and a plea to relax efforts in combating its ravages.

The antiseptic management of labour must form the keystone of obstetric practice and teaching, and there is still much scope for it to be more rigidly enforced.

Though, in spite of antiseptics, the mortality from puerperal fever remains the same as fifty years ago is difficult to believe, when we come to face the statistical figures of deaths from other puerperal diseases, and find a similar state of affairs, it becomes almost incredible. Is it that our up-to-date methods are mere fashionable millinery? or is it that woman in modern civilisation is becoming more fragile and degenerate? or that labours are more difficult from the increased size of the child's head—the result of intellectual development? Whatever it may be, statistical figures again show a similar condition of affairs. In Scotland, in 1855, the death-rate from other puerperal causes was 3·6 per thousand; in 1908, 3·3; while in Edinburgh it is shown to have increased.

On consulting Table III. (p. 30), this will be found to be due mainly to the great number of deaths from puerperal convulsions, which show an increase of nearly double.

Statistics are unreliable, and should not in any way be allowed to overcome methods which are based on a true scientific foundation, and, when adverse, should merely act as a greater stimulus to further development.

In England there is a marked drop from general puerperal diseases other than sepsis—from 2·9 per thousand to 2. This can be readily accounted for by the difference in their form of registration, which does not include under puerperal deaths pregnancy associated with Bright's disease, pneumonia, and heart affections, etc., which are all included in Scottish puerperal statistics. By their inclusion the mortality percentage would be raised to about the same as that of Scotland and Ireland.

The enormous increase of deaths from eclampsia in Scotland merits the closest attention. It has become a real scourge, and, so far, is apparently unpreventable.

According to the statistics of hospitals—

In Dublin Rotunda Hospital in four years—from 1906 to 1909 inclusive—there were 36 eclamptic cases, with 4 deaths, in 7845 cases of labour.

In Queen Charlotte's in four years—1906 to 1909 inclusive—there were 37 eclamptics, with 6 deaths, in 7000 cases.

In Edinburgh Maternity Hospital in four years—1906 to 1909 inclusive—there were 68 eclamptics, with 24 deaths, out of 2027 cases.

In comparing these we find the astonishing results that in Dublin and Queen Charlotte's the percentage of eclampsia is ·5 per cent., in which 14 per cent. were fatal, while in Edinburgh the percentage is 3 per cent., or six times greater, and of these 35 per cent. died, or 2½ times more fatal.

In St Mary's Hospital, Manchester, there were only 11 eclamptics in 4558 cases, or ·24 per cent. Of these, however, 6 died, or 63·6 per cent. This shows a still smaller percentage of cases, with a much increased mortality.

From these figures it is evident we in Scotland are specially prone to the development of this dread complication, not only in frequency but in potency.

Our methods of treatment are as up-to-date and in every way similar to those of other institutions, yet our death-rate is enormously higher. So far as the disease is concerned, we undoubtedly know more within recent years as regards its development from a physiological standpoint, yet from the aspect of prevention and treatment we are still hopelessly ignorant.

It is feasible to imagine that the marked increase in Scotland is due, perhaps, to the great difference in diet of the populace, who fifty years ago subsisted to a great extent on farinaceous food, instead of the highly nitrogenous feeding they now adopt. To this must be added the difference in climate, which, as has been frequently asserted, has apparently some effect on the development of eclampsia.

Knowing as we do, however, the extremely beneficent effects of dieting in albuminuria cases during pregnancy, a special effort should be made to try and instil into the minds of the masses the important necessity of having their urine tested in the later months of pregnancy as a routine. By this means alone, in the light of our present knowledge, can we hope to reduce the terrible mortality which exists from this complication of labour.

In considering, however, the sustained mortality from puerperal cases other than septicæmia, one cannot lay it entirely at the door of eclampsia, as we find on consulting Table III. (p. 30) that the mortality from placenta prævia, hæmorrhages, and other causes remains almost stationary. It is hard, indeed quite impossible, to believe that no real advance has been made in the successful management of complicated labour in the last fifty years, but, so far as the figures show, little tangible benefit has resulted.

If advance has been made, it must be compensated by a large increase in the number of complicated cases, which goes to show that so-called civilisation is tending towards extinction of the race, which is only maintained by improved methods of obstetric practice.

Look at it as we may, there is no obscuring the hard fact that one in two hundred women die in the so-called natural process of propagation of the race, in spite of our best-directed efforts on their behalf. The odds in favour of a woman having a family of five without losing her life are only forty to one. It would almost seem as if reproduction had passed from the physiological to the pathological.

Having dwelt thus shortly on the past progress of obstetrics and gynæcology, it is difficult to leave the subject without turning one's thoughts to the future and indulging in innocent speculation.

In this age of increasing specialism, it is needless to say that gynæcology has naturally become one of the most prominent specialities.

Though developed by the obstetrician, its essentially surgical character has naturally attracted the attention of the general surgeon, who, in many instances, has considered it a legitimate part of his calling, and accordingly annexed it. A state of guerilla warfare has therefore resulted over the ridge of the ilio-pectineal line, he (the surgeon) making incursions on the pelvis from the abdomen, on the pretext that an obstetrician is no trained surgeon, while, on the other hand, the gynæcologist, considering the surgeon was dealing with organs he knew nothing about, in the spirit of retaliation, pushed his campaign into the abdomen from the pelvis. Such a condition of affairs, though natural to expect in a new speciality, cannot continue to exist. For a time it may do so, but it seems to me a new speciality will eventually arise represented by the "abdominal surgeon."

It is, on the one hand, essential that the gynæcologist must be a thoroughly trained surgeon, while, on the other, the surgeon must be a thoroughly trained obstetrician.

It is absolutely necessary for the efficient and safe removal of uterine and fibroid tumours that the operator must be able to meet with any emergency, such as resection of the gut or the treatment of other abdominal conditions, for which the tumours may have been mistaken or otherwise complicated. Thus there seems no reason why the operator should not primarily operate for these conditions both in male and female.

In the same manner the operator must be thoroughly versed in the diagnosis of pelvic and other conditions, be able to eliminate pregnancy, and also be thoroughly conversant with the effects on pregnancy and labour of the operations he performs. He must not be a mere mechanic doing piecework on lines drafted by another, but have a thorough grasp of each individual case from both its physiological and pathological aspects.

The average general surgeon takes a pride in his ignorance of obstetrics; to the efficient abdominal surgeon of the future its knowledge must form a *sine qua non*.

It may be said that the so-called pure abdominal surgeon is a needless refinement of specialism already carried to excess! In this I do not agree. Specialism is only in its infancy, and is advancing in popular favour rapidly. It is the tendency of the age, not only in our, but in every other profession—the result of increasing knowledge—which makes it impossible for one individual to have a thorough grasp of every branch.

In my opinion the time is not far distant when, in large cities, there will be a complete change in medical practice. Specialists will be rampant in their various spheres merely as specialists, not as consultants. They will ply their calling for ordinary visiting fees, and will be employed directly without consultation, and will form a class of special practitioners

They will thus become much more accessible to the public, to the majority of whom consultants' fees are at present prohibitive, a state of affairs which probably accounts for the gross abuse of the special departments of our charitable institutions. That the general practitioner will become extinct I would not for a moment suggest, nor would I like to think. The family doctor never will be dispensed with; nothing could be more undesirable. Yet, from the increasing army of specialists working for similar fees, his numbers will be reduced, his assumed prescriptive right to attend the family under all circumstances removed, and thus medical etiquette rendered less involved.

Such a transformation may seem merely fanciful, but the drift of medical practice is strongly in this direction. Many of my older practitioner friends have told me of the rapid change that is occurring in their own experience, due to the inroads of specialism, especially among the well-to-do classes. That it is desirable is doubtful. The public, however, pay the money and call the tune. It is a subject full of importance and open to controversy; but I have already seriously digressed, and leave it for contemplation.

One other subject in this comparative review of obstetrics and gynæcology which merits consideration is the teaching.

In gynæcology, as is to be expected from its rapid development, many changes have occurred, particularly from its practical aspect; till now it forms a special practical part of the final examination.

Obstetrics, on the other hand, remains *in statu quo*, with the single exception that theoretically it has been divorced from gynæcology so far as the teachers are concerned. That this is desirable is very doubtful. As has already been pointed out, the abdominal surgeon or gynæcologist must be an expert obstetrician. The organs concerned, their method of examination, the origin of the pathological from the physiological, all go to blend midwifery and gynæcology so intimately together that

it is impossible that a thorough knowledge of the one can exist without an intimate acquaintance with the other.

To the gynæcologist the question of pregnancy ever asserts itself in his differential diagnosis—its presence, future possibility, or the exciting cause of existing conditions.

In other words, to alienate gynæcology from midwifery is to destroy its very existence and submerge it in the vast domain of surgery, which is a retrograde step.

It seems questionable, therefore, that students should be taught by different lecturers, however brilliant they may be, for on individual subjects their ideas may be different, and thus the continuity which is the essential of effective teaching is lost. Further, it tends to dissociate the intimate relationship of the two subjects in the pupil's mind.

The expert gynæcologist must be primarily an expert obstetrician, else the branch is broken from its parent stem. In actual practice it may be unworkable that the practice of abdominal surgeon or gynæcologist be combined with obstetrics; this is, however, merely a matter of practical convenience, and there is no valid reason why they should not be practised together except from this standpoint.

It seems to me that no one would be better fitted to manage complicated labours than the so-called abdominal surgeon, trained in the manner I have previously suggested. The methods of delivery in difficult labours are on the same platform as other manipulative surgical procedures, while the growing frequency of Cæsarean section, symphysiotomy, etc., render it essential that the expert obstetrician must be surgically trained.

Of much more importance, however, is the teaching of practical midwifery, which, unlike its sister branches of medicine and surgery, remains in essentially the same position it was thirty years ago. No advance has been made. The student is merely required to be present at a certain number of

so-called cliniques, and attend personally a given number of cases, or, as an alternative, attend personally 25 cases of labour.

True, the General Medical Council have proposed as an alternative, residence for a month in a lying-in institution, which is a step in the right direction, but, alas! this is not compulsory.

To all of us it may be assumed as an accepted fact that the only method of clinical instruction is seeing cases actually managed, be they medical, surgical, or obstetric. In the two former the greatest attention in this school has been given, that this should be acquired by the student. In midwifery, alas! it is not so.

The student is allowed by attending 25 probably normal cases to gain a certificate in practical midwifery. More than likely he may, in these cases, never see an abnormality. Even if he has added to this, attendance at so-called cliniques at given hours, his clinical knowledge of abnormalities is as prone to be equally deficient. To go round the puerperal wards of a hospital is entirely begging the question of true clinical instruction. Abnormal cases must be actually seen in progress and their management noted. It must be remembered that the essence of obstetric practice is to be able to conduct abnormal cases. If all confinements were normal, obstetrics as an art would cease to exist, and be practised by nurses and neighbours.

We as doctors are only paid to attend in case of accidents suddenly arising which demand prompt attention. How are students to be efficient in their management unless they have seen similar cases treated? Is it fair to turn them loose on the public with merely a book knowledge when clinical instruction can be had? The parade of the puerperal wards, so far as true clinical instruction in the management of labour is concerned, is a farce, and might be as thoroughly conducted at Madame

Tussaud's. They are merely of value clinically as a training in the management of the puerperium.

In the medical wards of the Infirmary, students are not shown patients who *had* pneumonia, but who are actually suffering. In like ways in the surgical wards they are not shown patients with stumps only, but see the actual amputations and follow their course after. Why should it be different in midwifery? Is it not more essential that he should be shown forceps applied or hæmorrhage arrested, which he may have to do without assistance the day after he qualifies?

Is attendance on 25 cases of normal labour, where he simply stands by, Micawber-like, of any value to the student in teaching him how to act in an emergency? Yet when he goes into practice he is engaged and paid by the public as experienced, in case of complication.

It is needless to say that the only method by which he can gain a clinical knowledge of the management of abnormal labour cases is by residence in a lying-in hospital, where not only is the labour seen, but the case followed during convalescence.

It has been argued that this is impossible: firstly, because there are not sufficient facilities; and secondly, that it will interfere with his lectures and other work.

In Edinburgh, fortunately, the first argument falls to the ground: the hospital facilities are ample for the clinical training of all. As regards the second, it seems to me that personal attendance on 25 cases will probably interfere more with his work both day and night than residence in a hospital for a month.

If our aim is to turn out efficiently trained obstetricians, there is but one way, and that is by hospital residence. It is bound to come in time if we as obstetricians are to keep pace with the improvements of clinical methods in medicine and surgery. It is already compulsory in one Scottish school—Aberdeen; and Glasgow and St Andrews have declared in

favour of it. Let us hope that Edinburgh, which has been so closely identified with the progress of obstetrics under the guidance of Simpson, Matthews Duncan, and others, shall be in the van by making it compulsory, and not be coerced into it after other schools have adopted it.

At the risk of further wearying you before concluding, one other subject might be touched upon, viz., the teaching and training of nurses and midwives.

At the present time the majority of women trained in Scottish Maternity Institutions take the qualifying certificate of the General Midwives Board as an advanced diploma, principally as a hall-mark of efficiency. For this they have to be examined in England, which has acquired the prescriptive right of granting it, on the plea that the Midwives Act applies to England only.

Further, the requirements of this Board are so exacting that it is impossible for nurses and students to be trained on the same cases, which very seriously handicaps the training of both in this and other Scottish schools, from the want of sufficient clinical material.

To obviate this unnecessary trouble on the part of women who desire a certificate of a general standard of efficiency, and allow of more use of our material being made, it would seem desirable that a Scottish diploma should be granted on similar lines.

There is, I believe, an active movement on foot at present in favour of extending the Midwives Act to Scotland. Should this be so, our endeavours must be directed to a special Act where the irksome and unnecessary demands of the English Act are so modified as to make it possible for our training of both students and midwives in our own hospitals.

This I consider of extreme importance, and must be at once energetically faced, otherwise we shall be left in a most unenviable position.

In having dwelt upon such a number of important and highly controversial subjects, I feel I have exercised my prerogative in addressing you to the full, and must claim your indulgence.

Should I have elicited any moot points of interest unknown to you, and stimulated thought towards their elucidation, I am proud. In all humility I have approached them, and I again thank you for having given me the opportunity of doing so by addressing you this evening.

[Tables I. to III. and Chart.

TABLE I.—*Puerperal Deaths*, 1855-1909.

Year.	Scotland.					Edinburgh				
	Estimated Number of Confinements.	Deaths from		Deaths per 1000 Confinements from		Estimated Number of Confinements.	Deaths from		Deaths per 1000 Confinements from	
		Puerperal Fever.	Other Puerperal Causes.	Puerperal Fever.	Other Puerperal Causes.		Puerperal Fever.	Other Puerperal Causes.	Puerperal Fever.	Other Puerperal Causes.
1855	96,850	169	341	1·7	3·6	4788	10	16	2·1	3·3
1856	105,556	143	351	1·4	3·3	5487	6	25	1·1	4·5
1857	107,186	145	304	1·4	2·8	5516	9	21	1·6	3·8
1858	107,858	152	302	1·4	2·8	5378	12	13	2·2	2·4
1859	110,445	175	333	1·6	3·0	5687	19	20	3·3	3·6
1860	109,553	236	328	2·2	2·9	5616	18	16	3·2	2·9
1861	111,010	203	308	1·8	2·8	5940	16	24	2·7	4·0
1862	111,042	130	305	1·2	2·7	5975	16	19	2·7	3·2
1863	113,465	195	376	1·7	3·3	6412	11	23	1·7	3·6
1864	116,526	254	374	2·2	3·2	6388	19	18	3·0	2·8
1865	117,272	213	422	1·8	3·6	6427	14	25	2·2	3·9
1866	117,891	181	356	1·5	3·1	6480	14	18	2·2	2·7
1867	118,285	163	321	1·4	2·7	6704	17	18	2·5	2·7
1868	119,743	140	354	1 2	2·9	6889	12	36	1·7	5·3
1869	117,513	153	410	1·3	3·5	7007	10	26	1·4	3·7
1870	119,687	202	381	1·7	3·2	6980	16	21	2·3	3·0
1871	120,535	225	420	1·9	3·5	7132	12	32	1·7	4·5
1872	123,228	219	391	1·8	3·2	6899	17	26	2·5	3·7
1873	124,173	251	325	2·0	2·6	7037	15	25	2·1	3·6
1874	128,282	378	442	2·9	3·5	7094	11	27	1·6	3·8
1875	128,187	389	459	3·0	3·6	7002	17	27	2·4	3·9
1876	131,283	231	374	1·8	2·8	7311	5	20	0·7	2·7
1877	131,637	163	394	1·2	3·0	7529	3	22	0·4	2·9
1878	131,651	164	380	1·2	2·9	7705	17	21	2·2	2·7
1879	130,450	184	397	1·4	3·1	7693	14	23	1·8	3·0
1880	129,272	185	416	1·4	3·2	7823	10	22	1·3	2·8
1881	130,963	235	462	1·8	3·5	7613	22	19	2·9	2·5
1882	130,965	267	403	2·0	3·1	7568	26	14	3·4	1·9
1883	129,205	363	427	2·8	3·3	7106	22	20	3·1	2·8
1884	134,045	336	371	2·5	2·8	7749	19	20	2·5	2·5
1885	130,881	374	405	2·9	3·1	7646	16	21	2·1	2·7
1886	132,866	270	331	2·0	2·5	7713	12	14	1·6	1·8
1887	129,199	275	338	2·1	2·6	7852	19	30	2·4	3·8
1888	127,958	309	351	2·4	2·8	7770	17	20	2·2	2·6
1889	127,407	277	346	2·2	2·7	7697	13	18	1·7	2·3
1890	126,083	324	363	2·6	2·8	7446	18	33	2·4	4·4
1891	130,815	378	340	2·9	2·6	7630	17	20	2·2	2·6
1892	129,718	315	373	2·4	2·9	7450	13	20	1·7	2·7
1893	131,902	252	336	1·9	2·6	7708	12	24	1·6	3·1
1894	129,036	285	342	2·2	2·7	7494	13	12	1·7	1·6
1895	131,384	253	355	1·9	2·7	7683	8	24	1·0	3·2
1896	134,019	220	357	1·6	2·7	7899	17	18	2·2	2·2
1897	133,846	205	331	1·5	2·5	8292	9	20	1·1	2·4
1898	135,852	227	351	1·7	2·6	8405	9	19	1·1	2·2
1899	135,702	214	341	1·6	2·5	8525	9	29	1·1	3·4
1900	136,318	225	342	1·7	2·5	8435	12	19	1·4	2·3
		S. P. F.*					S. P. F.*			
1901	137,150	118 4 158	347	2·0	2·6	8213	8 0 7	24	1·8	2·9
1902	137,127	106 3 198	375	2·2	2·8	8407	14 0 8	25	2·6	3·0
1903	138,489	110 6 175	418	2·1	3·0	8394	1 0 9	38	1·2	4·5
1904	137,512	113 2 126	374	1·8	2·7	8063	9 0 4	23	1·6	2·9
1905	136,245	124 5 119	450	1·8	3·5	8030	8 0 4	33	1·5	4·1
1906	136,961	123 10 130	442	1·9	3·3	7936	7 0 4	22	1·4	2·8
1907	133,492	135 6 87	451	1·7	3·4	7786	9 1 4	32	1·8	4·1
1908	136,140	107 3 121	435	1·7	3·3	7784	5 0 5	35	1·3	4·5
1909	...	...	...	...	...	7688	9 0 3	35	1·6	4·5

* S = Septicæmia and Sapræmia. P = Pyæmia. F = Fever.

Table II

Year.	Births Registered.	Estimated Confinements.	Deaths from		Deaths per 1000 Confinements from	
			Puerperal Fever.	Other Puerperal Diseases.	Puerperal Fever.	Other Puerperal Diseases.
ENGLAND AND WALES.						
1855	635,043	658,860	1079	1900	1·6	2·9
1860	684,048	709,703	987	2186	1·4	3·1
1865	748,069	776,125	1333	2490	1·7	3·2
1870	792,787	822,520	1492	2383	1·8	2·9
1875	859,697	882,509	2504	2560	2·8	2·9
1880	881,643	914,708	1659	1833	1·8	2·0
1885	894,270	927,809	2420	2029	2·6	2·2
1890	869,937	902,563	2016	2239	2·2	2·5
1895	922,291	956,881	1927	2292	2·0	2·4
1900	927,062	961,831	2017	2438	2·1	2·5
1905	929,293	964,116	1734	2171	1·8	2·3
1908	940,383	975,652	1395	1966	1·4	2·0
IRELAND.						
1865	144,970	150,407	284	644	1·9	4·3
1870	149,846	155,466	360	670	2·3	4·3
1875	138,320	143,508	442	563	3·1	3·9
1880	128,086	132,890	347	544	2·6	4·1
1885	115,951	120,300	370	495	3·1	4·1
1890	105,254	109,201	252	440	2·3	4·0
1895	106,113	110,093	321	448	2·9	4·1
1900	101,459	105,264	236	414	2·2	3·9
1905	102,832	106,689	227	346	2·1	3·2
1908	102,039	105,866	189	341	1·8	3·2
FINLAND.						
1870	63,748	66,139	...	535	...	8·1
1875	69,521	72,128	...	629	...	8·7
1880	74,784	77,589	...	654	...	8·4
1885	75,129	77,947	...	536	...	6·9
1890	77,860	80,780	...	460	...	5·7
1895	81,783	84,850	...	454	..	5·4
1900	86,339	89,577	...	427	...	4·8
1905	87,841	91,135	...	374	...	4·1
NORWAY.						
1870	50,434	52,325	144	...	2·8	...
1875	56,358	58,472	183	...	3·1	...
1880	58,923	61,133	147	...	2·4	...
1885	61,052	63,342	165	...	2·6	...
1890	60,108	62,362	159	...	2·5	...
1895	62,932	65,292	94	..	1·4	...
1900	66,149	68,630	111	73	1·6	1·1
1905	...	.	...	...	...	...

TABLE II.—*Continued.*

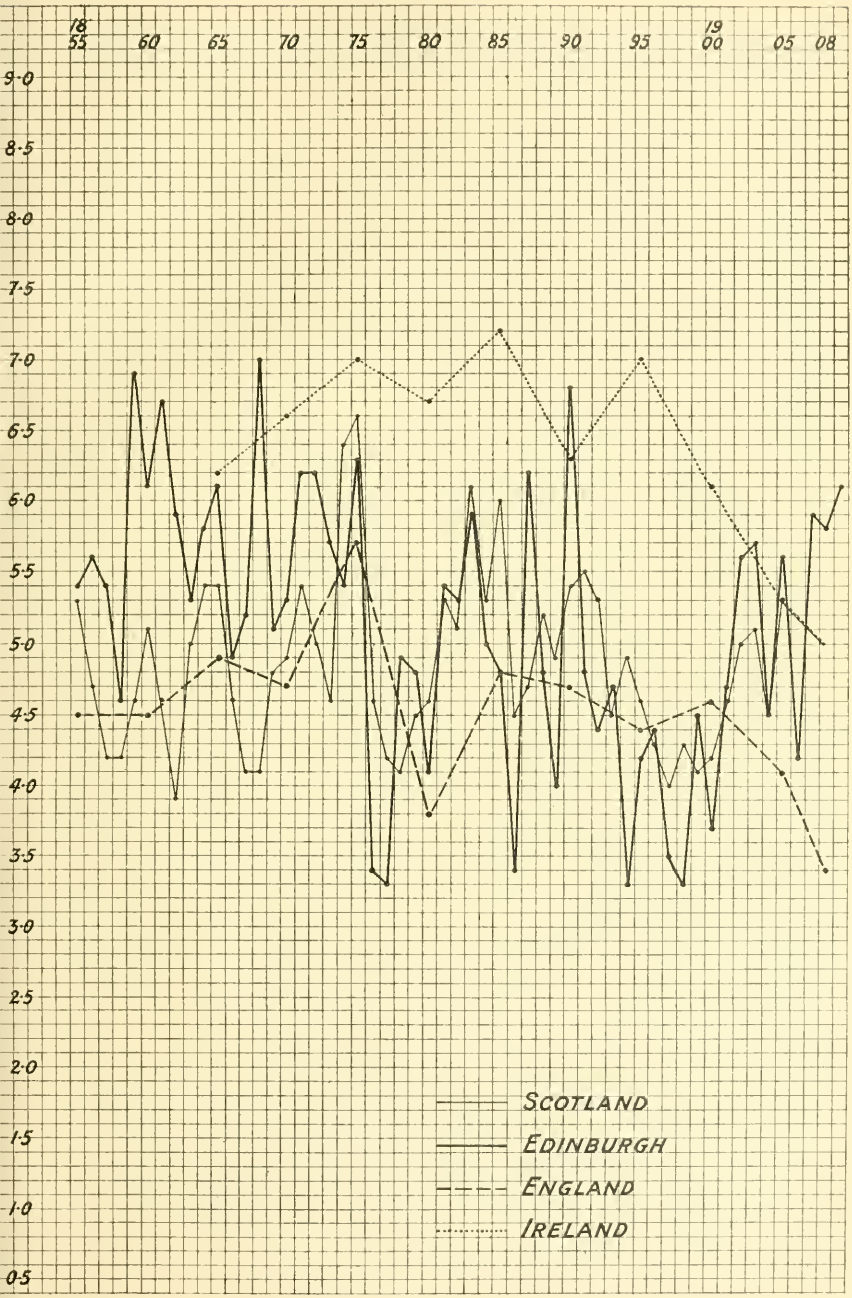
Year.	Births Registered.	Estimated Confinements.	Deaths from		Deaths per 1000 Confinements from	
			Puerperal Fever.	Other Puerperal Diseases.	Puerperal Fever.	Other Puerperal Diseases.
SWEDEN.						
1865	134,281	139,317	299	...	2·1	...
1870	119,838	124,332	380	...	3·1	...
1875	135,958	141,057	457	..	3·2	...
1880	134,262	139,297	338	...	2·4	...
1885	137,308	142,458	314	...	2·2	...
1890	133,597	138,607	173	...	1·2	...
1895	134,599	139,647	187	...	1·3	...
1900	138,139	143,320	121	...	0·8	...
1905	...	...	...	...	...	...
SWITZERLAND.						
1880	84,165	87,322	361	382	4·1	4·4
1885	80,349	83,362	436	309	5·2	3·7
1890	78,548	81,494	253	261	3·1	3·2
1895	84,973	88,160	217	281	2·5	3·2
1900	94,316	97,853	193	330	2·0	3·4
1905	94,653	98,203	253	...	2·6	...
HOLLAND.						
1875	138,469	143,662	235	651	1·6	4·5
1880	143,855	149,250	166	360	1·1	2·4
1885	148,028	153,580	214	463	1·4	3·0
1890	149,329	154,929	189	351	1·2	2·3
1895	158,130	164,061	154	328	0·9	2·0
1900	162,611	168,710	144	314	0·9	1·9
1905	170,767	177,172	119	295	0·7	1·7
ITALY.						
1887	1,152,906	1,196,145	2504	4436	2·1	3·7
1890	1,083,103	1,123,724	1682	2713	1·5	2·4
1895	1,092,102	1,133,061	1514	1893	1·3	1·7
1900	1,067,376	1,107,407	1033	2001	0·9	1·8
1905	1,084,518	1,125,192	977	2221	0·9	2·0

TABLE III.—*Scotland.*

Year.	Deaths from															
	Abortion, Miscarriage.		Puerperal Mania.		Puerperal Convulsions.		Placenta Praevia, Flooding.		Phlegmasia Dolens.		Other Accidents of Childbirth.	
1883	45	·34	27	·21	40	·31	83	·64	8	·06	224	1·73
1884	26	·19	17	·13	46	·34	84	·63	12	·09	186	1·39
1885	26	·20	26	·20	36	·28	94	·72	11	·08	212	1·62
1886	30	·23	14	·11	37	·28	79	·59	6	·05	165	1·24
1887	32	·25	15	·12	47	·36	76	·59	9	·07	159	1·23
1888	45	·35	18	·14	50	·39	79	·62	11	·09	148	1·16
1889	49	·38	15	·12	53	·42	78	·61	7	·05	141	1·13
1890	14	·35	13	·10	45	·36	75	·59	3	·02	183	1·45
1891	36	·28	14	·11	59	·45	80	·61	12	·09	139	1·06
1892	46	·35	22	·17	72	·56	85	·66	13	·10	135	1·04
1893	35	·27	14	·11	60	·45	64	·49	6	·05	157	1·19
1894	28	·22	17	·13	54	·42	86	·67	17	·13	140	1·08
1895	19	·14	20	·15	46	·35	84	·64	13	·10	173	1·32
1896	28	·21	7	·05	67	·50	67	·50	7	·05	181	1·35
1897	33	·25	10	·07	54	·40	82	·61	7	·05	145	1·08
1898	26	·19	6	·04	73	·54	88	·65	9	·07	149	1·10
1899	14	·10	6	·04	75	·55	106	·82	2	·01	138	1·02
1900	34	·25	9	·07	69	·51	79	·58	6	·04	145	1·06
1901	24	·17	10	·07	58	·42	81	·59	7	·05	167	1·22
1902	30	·22	11	·08	58	·42	60	·44	12	·09	204	1·49
1903	25	·18	9	·06	100	·72	94	·68	12	·09	178	1·29
1904	27	·20	10	·07	76	·55	86	·63	4	·03	171	1·24
1905	35	·26	9	·07	95	·70	101	·74	20	·15	210	1·54
1906	31	·23	7	·05	88	·64	86	·63	12	·09	230	1·68
1907	25	·19	6	·04	89	·67	91	·88	7	·05	210	1·80
1908	29	·21	10	·07	81	·59	81	·59	10	·07	234	1·72

Figures in heavy type show rate per 1000 confinements.

DEATH-RATE PER 1000 CONFINEMENTS FROM PUERPERAL CONDITIONS.



On the motion of *Sir Alexander Simpson*, seconded by *Sir Halliday Croom*, the President was accorded a hearty vote of thanks for the address.

V. *Mr Brewis* showed — DRAWINGS illustrating (1) two uteri with septic fibroids removed *post abortum* by supravaginal hysterectomy; (2) fibroid tumour of the uterus and tubal gestation removed from the same patient; (3) uterus removed by hysterectomy showing œdematous adenoma of the wall and adeno-carcinoma of the endometrium; (4) malignant adenoma of the ovary.

VI. *Dr A. H. F. Barbour* showed—(*a*) SARCOMA OF UTERUS; (*b*) DOUBLE PYOSALPINX.

VII. *Dr Haig Ferguson* showed—(*a*) INTERSTITIAL PREGNANCY. The patient, Mrs S., aged 23, had been married five months. She menstruated last on 1st of May 1910, and on the 24th of June was seized with severe pain and fainted, falling on to the floor. She was lifted on to the bed, and the doctor sent for. He prescribed powders, of which she took six, and remained in bed for three days. On getting up she felt very weak, and noticed that all her colour had gone. For a week, however, she was able to perform her household duties as usual. Then she went to friends near Aberdour, for a change. She felt much better, and her colour began to come back. On 15th July she walked 2 miles, and was afterwards seized with pain similar to the first attack, and sat doubled up by the roadside. She also had severe retching and vomiting. Her sister, who was with her, procured assistance, and she was carried home, half a mile across the fields, and put to bed. The pain got no better, and the vomiting persisted. Next day a doctor was sent for. He prescribed powders and brandy. The pain was not allayed, and continued, with vomiting, till

the 19th. On this date the sickness stopped, but the pain still continued. The doctor wished her to go to Dunfermline Cottage Hospital, but she preferred to go to her home in Edinburgh. On the 21st she travelled thence by train, having a 3 miles' journey by waggonette to the railway station. On her arrival home vomiting commenced once more, and she sent for her family doctor, who saw her at 7 P.M. and sent for Dr Haig Ferguson, who saw her at 8.30. She was admitted to Ward 36 at 9.45 P.M. The abdomen was opened the same night at 11 P.M., and was found to contain a large quantity of blood, fully 3 pints escaping. A tear was found in the uterine wall to the right of the fundus, and towards the posterior aspect. The fœtus, along with blood-clots and membranes, was removed. The uterus was then removed, by supravaginal amputation, along with the right ovary and tube. Before being closed, the abdominal cavity was filled with saline, and the patient was then put back to bed. The pulse-rate at this time was 160 per minute. Two pints of saline was infused under the mammæ, and the pulse-rate soon fell to 120, but remained rapid for some days—110 to 120. Recovery was uninterrupted, and the patient was discharged a month after operation.

(*b*) FIBROID TUMOUR showing myxomatous change. A. S., single, æt. 39. *Menstruation*—Twenty-eight day type, lasting two to three days, till four and a half years ago, since when it has steadily increased, coming on, during the last year, every two or three weeks, and lasting for ten days. On examination of the abdomen a large, rounded tumour, rather soft in consistency and reaching to the umbilicus, was discovered. It had been growing for four and a half years. Some difficulty was encountered at the operation in getting the tumour out of the pelvis. The operation was otherwise uneventful, and the patient made a complete recovery. During the time that the tumour was growing, great distress was sometimes experienced from attacks of asthma, which continued after admission, and

were so severe that the operation was not undertaken for nineteen days. Since operation, however, these symptoms have gradually improved, and when discharged the patient was practically free from chest symptoms.

(*c*) FIBROID showing red degeneration. Mrs M., æt. 34, had been married for six months, and was about three and a half months pregnant. *Menstruation*—Twenty-one day type, duration three days. No pain. Stopped three and a half months ago. For past two months patient complained of pain, sometimes severe, in lower abdomen. For past week pain was increasing so much that her doctor recommended admission to the Edinburgh Royal Infirmary. *Examination*—Immediately above the symphysis pubis, a little to right of middle line, a swelling, the size of a goose egg, was felt. *Operation*—The abdomen was opened on the 25th of July 1909. The uterus was found to be enlarged to the size of a three and a half months' pregnancy. A subperitoneal fibroid, the size of a goose egg, was found. It was adherent to the intestine, appendix, and omentum. Myomectomy and appendectomy were performed. The patient made a good recovery, and was dismissed on 16th August. A healthy child was born at the full term of pregnancy. The labour was normal, but there was some trouble from partial adhesion of the placenta.

VIII. *Dr Wm. Fordyce* showed—(*a*) TUBAL GESTATION which was associated with an intra-uterine pregnancy; (*b*) PREGNANT UTERUS removed by abdominal pan-hysterectomy for cancer of the cervix.

Meeting II.—December 14, 1910.

Dr F. W. N. Haultain, *President, in the Chair.*

I. The following gentlemen were elected Ordinary Fellows of the Society:—W. W. Carlow, M.B., Ch.B., 20 Eglinton Crescent, Edinburgh; John Michael Dewar, M.B., Ch.B., 24 Lauriston Place, Edinburgh; A. J. Gordon Hunter, M.B., Ch.B., St Catherine's, Linlithgow; W. Menzies Menzies, M.B., Ch.B., 20 Rutland Square, Edinburgh; W. P. Murray, M.B., Ch.B., 12 Merchiston Crescent, Edinburgh; David Shannon, M.B., Ch.B., 270 Bath Street, Glasgow; Lewis H. F. Thatcher, M.D., M.R.C.P.E., 8 Melville Crescent, Edinburgh.

II. *Dr Lamond Lackie* showed—(*a*) uterine fibroid with twisted pedicle, removed at the fifth month of pregnancy. The patient, æt. 32, had had one miscarriage about a year previously. When five months pregnant she was seized with sudden acute pain in the left side. Dr Mackintosh saw her and found a large tumour occupying the left side. It was thought to be an ovarian tumour, but turned out to be a fibroid. An operation was performed on the following day. On opening the abdomen acute inflammation was found, and on passing the hand down on the left side a tumour was felt with two twists on its pedicle. The patient did well and left the hospital at the end of four weeks; but within six weeks there was premature labour and the child was born dead.

(*b*) Molluscum fibrosum of external genitals. This specimen was interesting because it was practically unique. Dr Cranston Low had made a cast of the condition. The patient, æt. 57, was admitted to Leith Hospital some weeks ago. She had had six children and six miscarriages. The miscarriages were interesting as suggesting the syphilitic nature of the tumour; later on, however, it was found that there was no suspicion of syphilis. The condition began sixteen

years ago and gradually increased till twelve years ago, when it had attained the size shown in the cast. The patient thought it had not increased for twelve years. Twelve years ago her last child was born; since that time there had been no pregnancy. Dr Fordyce saw the patient and thought it was some sort of filarial growth, probably molluscum fibrosum, but the diagnosis was somewhat difficult. Dr Cranston Low diagnosed the condition as molluscum fibrosum. On other parts of the body—buttocks, face, trunk — there were similar fibrous growths. The possibility of elephantiasis suggested itself, but microscopical examination did not confirm this view, and the appearances were not such as are seen in that condition. Dr Cranston Low had kindly allowed him to show a cast of elephantiasis which had been brought before the Society fifteen years ago—a pure case of elephantiasis which probably was syphilitic. His own specimen had shrunk very much; it was removed by free excision. The cast really represented the appearance of the tumours at the time of the operation. Unfortunately the tissues were very sodden, and, in spite of great care, broke down. The patient did perfectly well for ten days, but after that she developed septic symptoms, and the sepsis spread to the kidney. She died on the fourteenth day of acute uræmia of septic origin.

(Two photographs taken when the patient was on the operating table, showing the condition at that time, were also handed round.)

III. *Mr E. W. Scott Carmichael* showed—(*a*) UTERINE FIBROID WITH HÆMATOMA OF THE OVARY, removed from a patient, æt. 49, who had consulted him for hæmorrhage of some weeks' duration. There was also great pain on the left side high up in the uterus. It looked like an inflamed ovary associated with a fibroid. The right ovary was found to be enlarged, and so firm in consistency as to suggest a gumma; on being opened,

however, it was found to be full of blood. The patient was an unmarried woman, and there was no possible suspicion of pregnancy. This case did not support the view that hæmatoma of the ovary was due to ovarian pregnancy. Hæmatomas were usually associated with fibroid tumours.

(*b*) DERMOID CYST ASSOCIATED WITH MALIGNANT DISEASE OF HEPATIC FLEXURE OF COLON.

(*c*) SUPPURATING OVARIAN CYST. This specimen was interesting because the patient was in hospital with a general peritonitis, and had been sent in as a case of appendicitis. It was impossible to be certain of the peritonitis because of the distension of the abdomen. On opening the abdomen it was found that an ovarian tumour had become infected by septic organisms, and had ruptured five weeks after labour. It was the patient's second child, and she had complained of no symptoms before.

(*d*) SEVERAL UTERINE FIBROIDS.

SOME CONSIDERATIONS REGARDING THE NARROW PELVIS.

By Professor Sir J. HALLIDAY CROOM, M.D., F.R.C.S.Ed., F.R.C.P.Ed.

OF late years the conditions concerned in delivery through a narrow pelvis have been considerably modified, and this certainly is the case in hospitals where septicæmia to a very great extent has ceased to be the nightmare of the obstetrician.

It must be at once frankly admitted that while septicæmia has in hospitals to a very great extent been modified and reduced to a minimum, yet in private practice, as has been shown to a demonstration over and over again, especially by the work of Cullingworth and others, it remains to a very great extent unchecked, and this entirely owing to the fact that antiseptics are not, and probably cannot be, observed to the same careful extent as they are in hospitals. And,

although maternity surgeons have seen and appreciated its benefits in hospitals, yet it is remarkable with what slowness great principles filter down from the hospitals to the profession, and antiseptic midwifery is not yet practised as it ought to be.

The same difficulty remains with regard to obstetric work proper. It is useless to discuss the methods of dealing with the narrow pelvis until the profession has recognised the fact that the first essential in dealing with such a pelvis is a proper estimate of it before labour sets in. For all treatment of narrow pelvis an early diagnosis is indispensable. It is evident that a safe choice of any procedure must depend upon an accurate estimate of the pelvis and of the fœtal head, and this means a knowledge of pelvimetry and abdominal palpation. Even then accuracy is only to be gained under certain limits.

In extreme cases of pelvic deformity diagnosis is self-evident, but until teachers devote more time to demonstrating the importance of early recognition of the disproportion between the head and the pelvis, and to the possible incidence of serious trouble, and till the practitioner makes it a rule to examine and measure pelves in all cases of primigravidæ, several weeks before the end of pregnancy, and to make during labour a careful estimate of existing conditions, no method of treatment can ever be expected to be satisfactory. This must of necessity be the foundation of all advances in obstetric art. Cases must come, and do come, to our maternity hospitals, too late for Cæsarean section or any other more accurate method to be safely adopted, and if we are ever to emerge from the despair of embryulcia, it will only be by a refining and improving of our diagnostic means.

In narrow pelvis as in all other conditions, preventive medicine is, after all, most valuable. That this early recognition and examination of the primigravid woman has been

shown to be of the utmost value in cases other than the narrow pelvis, is exceptionally well illustrated by the prematernity Hospital here. Into this institution are admitted primigravid women, at a comparatively early period of their pregnancy, and their condition is there examined very carefully. Although many of these women have shown indications of the toxæmia of pregnancy, yet, by careful prophylactic treatment, not one has been admitted to the Hospital where fulminating toxæmia as shown in eclampsia, has occurred, and in several the conditions of the pelvis have been early recognised, and appropriate methods of treatment adopted before labour actually set in.

If, as Schauta says, the general practitioner would get accustomed to recognise at the right time, degrees of narrow pelvis before the onset of labour, and assign all these cases early enough to obstetric institutions, then might progress be made toward the ideal treatment of contracted pelvis.

If it has taken nearly four decades for the general profession to absorb the great advantages of strict antiseptics in midwifery practice, one trembles to think how long it will be before the importance of making an accurate diagnosis in narrow pelvis will become an accepted fact. The day must surely come when perforation will be an operation to facilitate the delivery of a dead child only, and when perforation of a living child will become a relic of the past.

The general practitioner throughout the land is the accoucheur pre-eminent. To him we must look for the improvement from year to year in his aseptic technique, of the early recognition of abnormalities, and his improved conduct of the hygiene of pregnant women, and in the important conduct of delivery. Both maternal and fœtal mortality should be lowered, and can be. Far too many children's lives are sacrificed, and by forcible and unscientific deliveries. Far too many women and their unborn children

die from neglect and inattention to the various toxæmias of pregnancy, in their incipiency. Far too many women develop puerperal sepsis, and when discovered the treatment is neither modern nor effective. Every primipara without exception, and every multipara who gives a history of previous difficult labours, should be carefully examined by her attendant physician, as early as possible in her pregnancy, with the object of determining all that can be determined as to her general condition, her nourishment, her elimination, her probable conduct in labour, and particularly, as far as we are concerned to-night, with deformed or contracted pelvis.

The mortality for both mother and child in primary elected Cæsarean section is practically nil. When the operation is done later as the last resort, the mortality of the mother increases, but that of the child does not. (The statistics of pubiotomy would seem to show in this class of cases a little better chance for the mother, but not so favourable for the child.)

The principal points to obtain here are these: knowledge long enough prior to term to determine the size, shape, and capacity of the pelvis to go to term; then, to decide whether to adopt premature delivery at eight or eight and a half months, or to perform Cæsarean section, or to await the test of labour, and the condition such that the labour can be conducted aseptically. Premature delivery in contracted pelves at eight or eight and a half months is a procedure attended with good results, and a child so delivered stands as good a chance of living as at term.

Some writers have tried to devise means of determining the size of the fœtal head while *in utero*, but it is problematical if anything like an absolute estimate can be made under such conditions.

Let us now come to the question at issue, namely, the management of labour in a narrow pelvis.

With reference to the degrees of deformed pelvis, here I should like to say that I entirely agree with Dr Hastings Tweedy in respect to the measurement of these contracted pelves, and I should like to support his plea for their estimation in inches rather than centimetres, for whatever disadvantages the English method has in other respects, it certainly lends itself to a very simple and satisfactory division of pelvic contractions.

How, then, does the matter present itself?

Now, in looking at the management of labour in contracted pelves, one may at once place pelves in three categories. Those in which delivery of the living child *per vias naturales* is a practical impossibility—pelves measuring from 2 to 3 inches. Then, those in which delivery of a living child is possible with the aid of forceps, turning, or premature labour; and lastly, those cases in which spontaneous delivery may be with reason expected, namely, in pelves about 3½ inches. Now let us discuss the first and last, upon neither of which there can be very much room for difference of opinion.

With regard to the first set of cases, these are the cases which are simple to decide upon, namely, those where the child is alive and the true conjugate 2 to 3 inches.

These extreme degrees are easily recognised, and the management of such cases of extreme contraction, to my mind, resolves itself into simply the performance of Cæsarean section, which under the hands of a competent operator, and with reasonable surroundings, is practically safe, unless the patient has been handled and interfered with beforehand. Given a pregnancy at term, with a well-marked narrow pelvis, and where there has been no previous handling, Cæsarean section is obviously the operation by preference. Even in these extreme cases of pelvic deformity, if there has been handling beforehand, still, Cæsarean section of the Porro modification offers by far the best chance. To my mind Cæsarean section

stands out among all abdominal operations as the most satisfactory. In this operation all tissues and structures dealt with are healthy and normal. Nearly every complication can be foreseen and every danger prevented. There need scarcely be any mortality at all. The risks of Cæsarean section to the mother are very slight, and to the child still less, and its after effects, except for the possibility of ventral hernia, and abdominal and uterine scars and adhesions, are practically nil.

Obviously, with perfected Cæsarean section, the sacrificial operation of craniotomy must fall more and more into desuetude. With a pelvis so narrowed and recognised in sufficient time, there ought never to be recourse to sacrificial midwifery at all; but, as I have said already, until the importance of early diagnosis is recognised, there will be still a field, unfortunately, for craniotomy. That field is becoming more and more circumscribed every day, and, as I have said, in obstetrical institutions has almost disappeared.

Perforation really involves very little risk to the mother, because the mortality of craniotomy depends not on the craniotomy itself, but on the conditions for which it is employed. So it is that the mortality of craniotomy is greater in moderate contractions than in pronounced contractions, because in moderate contractions men were tempted to try forceps first, whereas in pronounced contractions they recognised at once that delivery of a living child by forceps was impossible. But the sphere of craniotomy must be very much limited unless the child be dead, and even then in extreme cases of deformity, dragging a mutilated child through a chink in the brim is by no means a desirable operation.

For the present, in private practice, until both the profession and the public have been educated past it, there must be a limited field for craniotomy.

Therefore, gentlemen, I am not one of those who go the length of saying that no living child should be perforated,

because there are unquestionably circumstances in which this is the only method of delivery to be seriously contemplated. In the present practice of midwifery, when the head has got caught at the brim, and forceps have been ineffectually used for a considerable time, where the parts have been bruised and possibly infected, what is there for the practitioner to do but craniotomy? Indeed, a condition like that really lends itself to very little else than craniotomy, and therefore, inasmuch as the mother's life and health are of greater value than the child's, perforation of a living child here is perfectly justified.

There is, however, another consideration as to the question of craniotomy, and it is this, that in private practice many practitioners shrink from the responsibility of performing Cæsarean section, not so much from their inability to perform the operation, as from their dislike of incurring the onus and the responsibility of suggesting what seems to the public a very serious and formidable interference. This, I think, will be an obstacle to the progress of all these operations to which I have to refer, in private practice, because, not unnaturally, such an operation in private practice is resented by the public, who prefer a mutilated child and an increased risk to the mother, to an operation that to them is surrounded with mystery and danger. In no sense can the use of craniotomy be called a scientific or satisfactory operation, and it must be strictly relegated to the position it deserves, of being taken only as a last resort.

But it cannot be too strongly emphasised that the disappearance of such operations as craniotomy and the conservation of fœtal life by other more appropriate methods, must, and can only, be accomplished by the more general adoption of scientific and careful measurement. So long as women are allowed to go into labour without the condition of the pelvis being ascertained, so long will infantile mortality remain high. And, conversely, with systematic examination of the pelvis, and the early recog-

nition of any defect, so, by means of comparatively recent advances in midwifery will fœtal life be spared.

But it must always be distinctly borne in mind that in dealing with the statistics of all obstetric operations and interferences, we are dealing with those only of Maternities and Hospital Cliniques, where all means and appliances are at the operator's hands, and where he has no one, but probably his colleagues, to interfere with his lines of action. His decision is final, and whatever his judgment may decide upon, and whatever the issue may be, there is no room for discussion or regrets. He is the final court of appeal. Obviously it is quite different in private practice, which after all, forms the bulk of obstetric practice, in this country at least.

Here statistics are to a great extent unavailable, and here the practitioner is handicapped by varying and often unsatisfactory surroundings, and by interference of friends, and he is obliged to do what is easiest for the mother, whatever be the result to the child.

It is further a fact, that, unlike in medicine and surgery, a consultation in pure practical obstetrics is very seldom requisitioned. In practical obstetrics every practioner is a law unto himself, and considers it rather a loss of prestige to be obliged to ask the advice of a Maternity surgeon in a bad case of labour.

Again, till it is a recognised fact that all important operations should be performed in a Home or Hospital, no very great advantage will be secured. Not many years ago a battle was fought against the system of performing abdominal operations in private houses, and abdominal surgery has become almost perfected. The same applies to midwifery. The problem of difficult operative midwifery will never be solved until sepsis is avoided, and all pregnant women who are threatened with serious complications, are taken to a Home or Hospital for the purpose of delivery. It is remarkable—though, of course, not

very difficult of explanation—what an objection women have to this. I think it is incumbent upon all of us who are teachers to emphasise the fact that only in this way will the mortality and morbidity of serious midwifery be reduced.

Thus far there can be very little doubt as to the method of dealing with these degrees of contraction. That is to say, any contraction between two and three-fifths and three and three-fifths. Given an accurate diagnosis, Cæsarean section is the treatment. But the question is altogether different when we approach the question of minor deformity. Let us pass from these extreme degrees of contraction to those slighter forms where accumulated experience has shown that spontaneous delivery may be with reasonable confidence expected. Hitherto in the management of such cases the most popular forms of treatment had been forceps, turning, and induction of premature labour,—that is to say, a conjugata vera of from three and a half to three and three-quarters.

Such operations as turning, premature labour and forceps, were had recourse to unnecessarily, because these methods of delivery were based upon two principles: First, the fear of septicæmia, and secondly, a too hard and fast law as to the danger of delayed labour and of the inability of Nature to overcome the difficulty. But, with the disappearance (in Hospitals at least) of puerperal septicæmia, confidence in the forces of Nature has returned, and labour complicated with a narrow pelvis is allowed to complete itself spontaneously; and this expectant method of treatment of labour in a narrow pelvis, if I may so call it, has proved satisfactory, and many patients who had previously been delivered by the aid of high forceps, version, and induction of premature labour, have succeeded in delivering themselves spontaneously.

Though with us, until recently, the second stage of labour was arbitrarily limited by time, now in view of the results obtained at home, and especially abroad by Pinard, Saxtorph,

Zweifel, and Dœderlein, we must modify our opinions, because they—not to mention many others—have shown that 80 per cent. of women with narrow pelvis of the minor degree, of which we are now speaking, deliver themselves spontaneously. Take for example the statistics culled from that excellent paper of Dr Little of Montreal. We find that in 87 contracted pelves there were 70 normal labours, and only 17 cases of dystocia. That is, 80 per cent. of these labours ended without interference. And not only was this the case, but the difficulties encountered were rarely great, Cæsarean section being performed only once for pelvic deformity. Again, for example, in Schauta's clinique, in 5228 cases of contracted pelvis, among 49,397 births, spontaneous delivery occurred in 4116, or 77 per cent., with a maternal mortality of ·09 per cent. and a fœtal mortality of 4·1 per cent., which is a result to the fœtus better than that given by any method of treatment with the exception of Cæsarean section. Buerger of Vienna reports 76 per cent. of spontaneous deliveries out of 4600 cases complicated with narrow pelvis.

Nothing, I think, is more striking in regard to this question of spontaneous delivery in narrow pelvis, than the low fœtal mortality. Boeninghausen, quoted by Kerr, points out that the fœtal mortality for spontaneous delivery in generally contracted pelves is ·2 per cent., and for flat rachitic pelves 2·7 per cent, while in artificial termination labours the mortality was 41 per cent. for generally contracted pelves, and 47 per cent. for flat pelves. This certainly is very striking, and is a commentary on the teaching that used to obtain in this country, where a definite time limit was laid down for the second stage of labour.

In view of the spontaneous delivery in narrow pelvis, the amount of cases requiring artificial aid must, therefore, become very small.

What are the limits of pelvic contraction that admit of spontaneous delivery in narrow pelvis?

In degrees of contraction slightly under 3½ inches for flat,

and $3\frac{3}{4}$ inches for generally contracted pelves, spontaneous delivery may be readily looked for.

With the knowledge we now possess that spontaneous delivery does occur in these cases, our obvious duty is to trust to the natural efforts, aided in many cases, by maintaining the patient in the Walcher position.

This Walcher position should never be omitted or neglected as an aid to the fixation of the head. This position may be maintained indefinitely by placing a patient in the cross-bed position, with her hips elevated by raising the side of the bed on blocks. This enables the patient's legs to hang down without permitting the feet to touch the floor. Two chairs are placed by the side of the bed, and the patient, covered with blankets, is instructed to let her legs rest on them in the intervals. During the uterine contraction, the legs hang down.

Now, in the lesser degrees of contraction, it is not unusual to find eight or ten hours occupied in moulding the head. While, until recent years, most teachers used to decry waiting during the second stage until fœtal or maternal signs of distress manifest themselves, now, in these narrow pelves, I think that the duration of the second stage of labour cannot be fixed by any artificial time limit, but only by watching the progress and the effect on both the mother and child, and leaving a sufficient length of time in the second stage for engagement and moulding. Naturally it must be kept in view that in these narrow pelves early rupture is frequent, and consequently slow dilatation of the cervix. When such is the case, labour can be very considerably expedited by simple dilatation of the cervix after the manner of Harris, with strict antiseptic precautions.

And now, having disposed of the upper and lower limits of the pelvis, we are left to decide what is to be done in the intermediate quarter of an inch that still remains to be dealt with. And here, obviously, is the field in which the greatest diversity of opinion exists.

What are the possibilities in such a pelvis?

We have a choice of operations. If the condition of the pelvis be recognised sufficiently early, we have the option of premature labour; if the case be at term, or in labour, we have the option of delivery by means of the forceps, or by some operation for enlarging the pelvis.

Now, what is the position exactly of the induction of premature labour?

I venture to say that if one takes up the statistics of any hospital with regard to the induction of premature labour, the safety to the mother is at once striking, as compared to the risk to the child, which is unfortunately very high. The only difficulty lies in selecting the proper time to perform this operation; but with a due regard to pelvic measurements and the adoption of Muller's technique, which must be conducted very carefully, it is an operation which is of much value, and does not deserve to be ruthlessly pushed aside as some foreign obstetricians would have us believe.

After all, the pelvic measurements are only one factor in the matter. The undue size of the child's head, or the early ossification of the child's head, is also a factor to be kept in view, and certainly the most difficult factor of the two to be estimated accurately.

Let me look shortly at the statistics of this operation in my own hospital first of all.

In looking over the statistics of the Edinburgh Hospital during the last two decades, I find that the induction of labour has been performed 61 times for contracted pelvis, and as the result of these 61 inductions, one mother (1½ per cent.) has died, and that death was apart altogether from any obstetric complication, because she died from advanced heart disease and emphysema. But, contrast that with the infantile mortality, and we find that of the children 33 out of 61 were dead. That is to say, 54 per cent.

It is not quite fair to take the Edinburgh Hospital as an exhibition of the fœtal death-rate, because it is a comparatively small hospital, and a large preponderance of serious cases are admitted to it. Many other hospitals—Queen Charlotte's, for example—have better fœtal results. Take last year's Report of the Queen Charlotte Hospital. In 36 cases of labour induced in a contracted pelvis, the fœtal mortality was only 14 per cent., and the maternal mortality nil.

In a very careful and remarkably interesting paper, Dr Blacker gives the statistics of the University College Hospital during the years 1897-1908. With contracted pelves treated by induction of premature labour, of 93 cases, 64 children were born alive and 29 were born dead, a mortality of 31 per cent.

I will not weary you with statistics, but I feel confident that taking the average of various institutions where the reports are accurately given, one may take it that the fœtal mortality attending premature labour is on the average about 30 per cent. And this fœtal mortality is very considerably increased if we consider the fact that quite a number of these premature children die within the first year, so that we have here (in premature labour) a method of dealing with a deformed pelvis with the utmost safety to the woman, but with unfortunate results to the child.

If, then, induction of premature labour be an operation which is practically safe for the mother, but of serious import to the child, what alternatives present themselves to us, because after all, the object of the obstetrician is to save both the mother and the child, and it ought to be our object to reach that ideal? Can we offer a woman any interference as safe as the induction to herself and with increased safety to the child? Our choice must lie between Cæsarean section at the close of pregnancy, and before labour sets in, or, secondly, the performance of pubiotomy or symphyseotomy, or the intra-pelvic method of application of forceps and subsequent pubiotomy if required.

Those operations are undertaken mainly with a view to improve the prospects of the child, and they can only be accepted if, while improving the fœtal mortality, they add no greater risk to the mother.

The possibilities of Cæsarean section and its mortality have been already discussed, and we now approach the subject of the operations for enlarging the pelvis, and of high forceps.

With regard to high forceps in narrow pelvis, the mortality and morbidity to the mother is unquestionable. Without entering into inevitable statistics, I believe that we may safely admit that the maternal mortality, even where the cases are quite aseptic, remains about 4 per cent., whereas the fœtal mortality runs between 30 per cent. and 40 per cent. No one has shown this better than Mr Kerr, who has pointed out exactly that according to the diminution in the pelvis in or near the area to which I am now referring, the fœtal mortality varies from 15 to 46.

Now, as against this fœtal mortality in induction and the mortality and morbidity to the mother in high forceps, we have operations for the enlargement of the pelvis suggested —practically symphyseotomy and hebosteotomy. These operations have had a somewhat chequered career, and it is only within the last two decades that they have been brought prominently and seriously forward. They have enthusiastic supporters in Germany, France, and Italy, but they have met with but very scant support in America and Canada, notwithstanding the powerful advocacy of Whitridge Williams, who performed 13 operations with success, as far as the mother was concerned, but with 3 fœtal deaths — which after all, amounts to 23 per cent. But perhaps Dœderlein's reports have done more to bring this method of delivery forward than any other.

Dœderlein gives 294 cases. In 77 of them the open method (Gigli) was followed. Eight mothers died (10 per

cent.). In the other 217 cases, he pursued his own methods, and 9 mothers died (4 per cent.).

On the other hand, although this represents the best that possibly can be done, the mortality to the children yet remains about 7 per cent. This represents the best results under the hands of the most experienced specialists in Germany and France, such as Dœderlein, Zweifel, Pinard, Krönig, Bumm, and Baisch, and it is but reasonable to expect that with increased experience, the conditions will be still more favourable.

Considering the serious possible risks of these operations such as hæmorrhage, tears, thrombosis, injuries to the bladder, and so on, it must be kept in view that even those are operations which for their successful performance must be conducted in an operating theatre of a Home, or in a Hospital. Even there, their sphere must be very limited.

In Dœderlein's clinique, premature labour, high forceps, and version have been discarded, and his clinique points with pride to the wonderful results obtained by themselves, Zweifel, Pinard, Krönig, and others, by this comparatively new treatment of narrow pelvis.

There can be no doubt that, at the last Gynæcological Congress in Dresden, Dœderlein's success with hebosteotomy caused a profound impression. The belief seems to be growing in Germany that the old-established treatments of narrow pelvis will become sooner or later obsolete, and that subcutaneous pubiotomy will supplant them.

I am not concerned with the advantages of hebosteotomy over symphyseotomy — that is a matter of detail, and as far as my knowledge and experience is concerned, I think symphyseotomy is the simpler operation of the two. Subcutaneous symphyseotomy can require but very little more than a tenotomy knife, and can be finished in a minute or two.

In order that these operations may be successfully carried

out, four obvious essentials are necessary. The child must be alive; the contraction must not be beneath 3 inches; the passages must not be infected; they must be well dilated. Further, though this is not conceded by all authorities, yet it seems to me, that one of the essential features in the successful performance of symphyseotomy is, that after the operation has been performed, the delivery of the child should be, so far as possible, spontaneous.

This opinion, I know, is traversed by Whitridge Williams and others; still, if the section of the pelvis admits of the enlargement to which I have already referred, spontaneous delivery should be possible, and should diminish the risks of artificial interference. In a very recent paper I observe that Pankow, writing from Krönig's clinique, states that the operations should never be performed in primiparæ, and in multiparæ only where there is reason to expect rapid and spontaneous delivery afterwards. Given these conditions present, what may we expect from the adoption of these methods?

In this country they have been performed successfully in England, Scotland, and Ireland, but of course, to a very limited extent as compared to their great frequency on the Continent.

Look at the statistics a little more carefully. It may be at once granted that the results on the whole are satisfactory. Take, for example, Munro Kerr's table of symphyseotomy:—

	No. of Cases.	Maternal Deaths.	Fœtal Deaths.
Bar . . .	23	0	6
Pinard . .	100	12	12
Zweifel . .	12	0	0
Herman . .	8	0	0
Munro Kerr .	9	0	0
Buist . .	8	0	2

With our present knowledge and experience, what then is the position of these operations on the pelvis?

To me it appears there never can be a question of selection, because, given a primipara whose pelvis has been carefully measured in ample time, then the option lies between induction and Cæsarean section (and probably in a primipara Cæsarean section is preferable).

On the other hand, take a multipara who has had a child sacrificed in her first labour, or induction with a dead child. Then obviously on the next occasion the election would lie not with a hebosteotomy or symphyseotomy, but rather with a Cæsarean section if taken in ample time. Or, possibly, she should have the option a second time of induction more accurately guaged and carefully performed. Pubiotomy as far as it has gone yet, certainly does not uniformly save the children, and it is not unassociated with grave maternal risk and death, whereas Cæsarean section, if undertaken in ample time, invariably saves the child, and the mother's risk is but slight.

Even if undertaken when labour is in progress, Cæsarean section still offers a good chance, and even when the passages have become infected, Cæsarean section, if followed by hysterectomy, is an undertaking to be considered. Where, then, do symphyseotomy and hebosteotomy stand?

Now, the case in which hebosteotomy and forceps compete, or where the one is the complement of the other, is where with moderate contraction the head is unengaged, and where ample time has been given for the head to enter. Then, obviously, forceps is not a desirable operation, because the grasp of the forceps can never be an accurate one, and extreme traction of the unmoulded head through the narrow brim is a very serious source of additional risk. There pubiotomy would compare distinctly favourably with forceps. If the mortality of forceps at 3 inches be 40 per cent., and

in the contraction to which we are alluding at present, something between 23 per cent. and 15 per cent.; and if these operations have a mortality to the child of only 7 per cent., it is obvious that this is the operation of preference there.

Then, again, symphyseotomy may be a most valuable complement to high forceps, because where forceps have been used, and where after reasonable use of forceps, no progress is made, then the operation of pubiotomy or of symphyseotomy may obviate a craniotomy. At the same time it must be remembered that the application of forceps after section of the pelvis, is only justified by the existence of danger to the child.

It seems to me, therefore, that the position of those operations for enlargement of the pelvis resolves itself into this, that they are to be undertaken as a matter of option, with the unengaged head at the brim. Indeed, no high forceps operation should be seriously undertaken unless the operator is prepared to perform one of these cutting operations in the event of the forceps operation being unsatisfactory. Thus, it seems to me, we have the operations of induction of labour, Cæsarean section, high forceps, craniotomy, and the enlargement of the pelvis put on a more or less satisfactory basis, without going to the extreme measures advocated by the foreign operators.

But let me for a moment hark back to the subject of Maternity Homes, which will, in the near future, be necessary. Gentlemen, if you send your patient into a Home or a Hospital for an amputation or a hernia, surely there can be no sufficient reason why an obstetric patient should not have the same advantages.

Gentlemen, would any of you by preference perform a hysterectomy or an ovariotomy in private? I myself have done two thousand of these operations, and I do not think half a dozen have been done in private. They have all been done

in Hospitals or Homes. Again, I ask you the question, if that be the safety in these gynæcological operations, why should the same safety not be accorded to obstetric operations? Surely the Cæsarean section is a simple enough operation to those of us who are engaged in obstetric work, yet anyone prefers to do this operation in a Home rather than in a private house.

Dr Munro Kerr said he had come specially to hear what Sir Halliday Croom had to say on this very important subject. He thought the two important points which had been brought out were:—First—What could be done by leaving the case to Nature? That was a point which had only come to be fully appreciated during the last few years. As a matter of fact, he (Dr Munro Kerr) had intended to write and ask the Secretary if he might be allowed to read a paper on similar cases to Sir Halliday's; he felt almost compelled to do so, because in the Edinburgh school axis-traction forceps had been so strongly advocated he was afraid it had not been recognised that high forceps must be used with great discretion. The last time he had heard the subject discussed was at Carlisle, where Dr Milne Murray opened a discussion on contracted pelvis; and the feature of his remarks was that he adopted an attitude of boastfulness as to what he could do with high forceps; if he remembered rightly, Dr Milne Murray said he had pulled a child through a conjugate of $2\frac{7}{8}$. He (Dr Munro Kerr) used to pull children through also—he used to put his foot on the bed and pull the child through. But that should not be done; it was not obstetrics. With that treatment he found he had a fœtal mortality of about 50 per cent. Of recent years, giving endless time, he had found that in a surprising number of cases the birth took place spontaneously—nothing was more striking than that fact, and it was being proclaimed from the cliniques of this and other countries.

The second point was that he entirely agreed with Sir Halliday Croom regarding the place of pubiotomy. Pubiotomy was a kind of "push" operation, a little bit that one keeps up one's sleeve. The head is estimated carefully, and after the case has gone for endless time and it is found that unless one uses brute force the head cannot get through, then one says to oneself "I will do pubiotomy." It is not scientific to say I will choose this case or that for pubiotomy; that is a wrong attitude, and one which Pinard himself had given up—he had gone back to Cæsarean section. He thought they were apt to attach too much importance to continental authorities, who were given to fashions. Obstetrics in Great Britain had never been of that kind, either in London or Edinburgh. At present the fashion in German schools was extra-peritoneal Cæsarean section; but he felt quite certain that in a couple of years nothing would be heard of it. He agreed with almost everything that Sir Halliday had advocated in the treatment of contracted pelvis.

Dr J. W. Ballantyne said he had listened with great interest to Sir Halliday Croom's paper, and was in almost perfect agreement with him. He found it very difficult to get an early examination in pregnancy, especially in primiparæ. He had had to do craniotomy in primiparæ, or where no knowledge of the pelvic condition had been gained from the first or even the second labour. He had said to doctors: "It is a pity you had not known of this a little earlier." They had answered: "We are not engaged during pregnancy, we sometimes are not engaged until the end of the first stage, and therefore it is impossible to know when we are dealing with a contracted pelvis! He thought it was very important to educate women—by means of nurses, etc.—as to the risks in contracted pelvis, and the importance of diagnosing the condition early in pregnancy. That was one line of advance which he thought the future would show. He also agreed as

regards the advantage of treating patients in Homes and pre-maternity wards; he thought that the hospital treatment of pregnancy would be another advance in the future. The reference to the Walcher position suggested to him that they might have a "labour" bed. Surely there ought to be something better in the Edinburgh Maternity Hospital than the ordinary bed, something which would allow of the Walcher and other positions being maintained. He did not propose a complicated operating table, but certainly a table which could be more easily managed than the ones they had at present. There was room for an obstetric operating table.

Dr Haig Ferguson said Sir Halliday Croom's paper was a very practical one. It was interesting to look back on the complete change which had occurred in our views as regards high forceps. He did not think that anyone of experience would now consider that forceps on the unengaged head was a justifiable operation, either for the sake of the child or the mother, and one saw most remarkable results (now that they had got rid of the bugbear, which used to be taught, that a woman's second stage was never to last longer than three or four hours) by giving plenty of time and trusting to Nature. He did not go the length of Dr Munro Kerr and wait an endless time for the head to come through, but certainly by giving plenty of time it was extraordinary how the head moulded through; afterwards the application of forceps was perfectly safe both for the mother and child, and in many cases forceps are not required. The other point which had struck him was that of induction of premature labour, and with Sir Halliday's remarks he most cordially agreed. Regarding the statistics of hospitals, he thought there was a fallacy with regard to the fœtal mortality, because in the case of hospitals women did not usually come in until they were very near their full time. Now the induction of premature labour performed then undoubtedly made it a little easier for the mother, and it was not

surprising that the maternal mortality was practically nil; but it does not help much as regards the child; therefore the mortality from induction was very much greater in hospital practice than it was in private practice, where one had the opportunity of watching the patients during the last few months of pregnancy, of measuring the pelvis and fœtal head, and, as it were, stepping in at the psychological moment. He thought Sir Halliday's paper of great importance, as it reflected the modern teaching and practice of obstetrics.

Dr Michael Dewar asked Sir Halliday Croom if he would in a word say what was the very minimum conjugate he would allow the general practitioner to apply forceps at full time; he asked it for his own guidance.

Sir Halliday Croom answered that 3½ inches, even 3¼ inches admitted of spontaneous delivery, so that forceps should not be used under 3¼ inches; not at 3 inches, as was sometimes advised.

Dr James Ritchie said it was of great importance to know beforehand the size and shape of the pelvis. He did not agree with Dr Munro Kerr that one ought to give unlimited time. His rule had been to give a very long time, especially in primiparæ, and so long as advance was being made and the patient's strength maintained, not to interfere. He had listened with great interest to Sir Halliday Croom's paper.

Dr Fordyce said there was one operation he was sorry Sir Halliday had passed over somewhat lightly, namely, turning. He thought they were losing sight of the old operation of turning. He did not exactly know what was meant nowadays by "high forceps." In the old high forceps cases, where the head was really above the brim and forceps were applied, he was quite sure better results would have been obtained if turning had been done. Many patients could be delivered in this way of a living child where otherwise the child would be born dead. He had seen cases where there had been one labour

and the child was born dead, and subsequently by turning he got a living child. He had come across doctors in the country lately who had found that by turning they had been successful after the forceps operation had failed. It was an exceedingly difficult thing indeed to estimate exactly the conjugate diameter of the pelvis—the abdomen had to be examined, the diagonal conjugate written down—it was difficult to be sure of $\frac{1}{2}$ to $\frac{3}{4}$ of an inch—and also the position of the head had to be taken into account (R.O.P. was more difficult than L.O.A.). Given a fat primipara with a rigid abdominal wall, it was not so easy as some people would make out to tell that the head was ready to engage in the brim or that it could be pressed into the brim. If, on being pushed downwards, it could not be made to enter the brim, then it was time to induce premature labour.

The President said he had enjoyed Sir Halliday Croom's paper extremely. He would like, however, to put in a word for the forceps; it seemed to him that they had been too much condemned by previous speakers. If there was no special indication against their employment, forceps should be used when labour became too lingering. To say that 4 per cent. of women died from high forceps seemed to him inconceivable in the hands of an efficient operator. He had used forceps at the brim in many cases, and had seen them used still more frequently, and he did not think he had ever seen one woman die as the result. One has to ask the question—When are we to apply forceps—what working rule are you going to give the general practitioner or student, as to when he is to apply them? Dr Munro Kerr said to wait an endless time; that he thought was ridiculous. What are the points by which you know when to put forceps on? He agreed with Dr Fordyce that it was a difficult thing to measure the pelvis; also to judge the extent of ossification of the head, and the amount of moulding it would undergo. If, after making out all these points to one's satisfaction, one still found the head not

engaging or not descending, what is one to do? Must one wait for a brown discharge, or a high temperature, or what? There seemed to him to be a middle line as regards the use of forceps; he though the pendulum had swung too far in the other direction. There were several other things he would have liked to say about this paper, which was one of great practical importance, but there was another important paper to be read and time was going on.

Sir Halliday Croom in replying said he would like to draw Dr Ballantyne's attention to the fact that in Sweden they had an almanack, published at sixpence and widely distributed. In every number there was an article giving information on some medical point, and in the last were some instructions to women (Dr Ballantyne, interrupting, said his point was how to get the paper into the hands of women.) Sir Halliday did not agree with Dr Fordyce as to the difficulty of measuring the pelvis; the size, he thought, could be approximated very nearly by taking the internal and external measurements. With regard to turning, his experience had been very unsatisfactory; he had had more unfortunate results from turning than from forceps. He thanked Dr Haig Ferguson for his remarks. He thought the President had misunderstood his attitude as regards the forceps; he had no antipathy to the forceps, far from it; he thought they were one of the best instruments they had in their possession. But to pull the child's head through a narrow brim and injure the mother was a different thing altogether. He still adhered to the view that the forceps were very essential in their proper place; but when they failed to do their duty, one of the operations which he had mentioned should be resorted to. He could not agree with the President; he maintained that women were frequently injured by forceps and children often lost.

V. THE STRUCTURE AND FUNCTIONS OF THE UTERINE MUCOSA; THE NATURE OF THE CHORIONIC ACTION AND THE FUNCTION OF THE DECIDUA. (WITH LANTERN DEMONSTRATION.)

By James Young, M.D., F.R.C.S.Ed., Gynæcologist to St Saviour's Dispensary; Physician to Lauriston Pre-Maternity Home.

Structure of the Uterine Mucosa and the Cause of the Opening-up of the Vessels during Menstruation.

To understand the manner in which the maternal tissues react to the stimulus of the engrafted ovum, a clear conception of the minute structural features of the uterine mucosa, especially of the vessels and the interglandular connective tissue or stroma, is essential. In the resting state the stroma consists of a mass of cells densely packed together and intimately united with their neighbours by means of protoplasmic strands. This description is the one found in the text-books. The blood-vessels, with which the mucosa is especially richly supplied, have a flattened intima, and, in vessels thicker than capillaries, a varying number of layers of concentrically arranged supporting elements.

But this description is incomplete; a more intimate acquaintance with the finer structure is necessary. As I have elsewhere shown, this is obtained by a study of the changes which occur in menstruation. In the premenstrual phase the mucosa is thickened chiefly by a free œdematous escape into the stroma, the cells of which are spread apart. There is also some blood-escape in the proximity of the vessels, which are often markedly expanded. As usually described, this fluid- and blood-escape is dependent upon the fact that before and during menstruation there occurs a congestion of the uterine vessels,

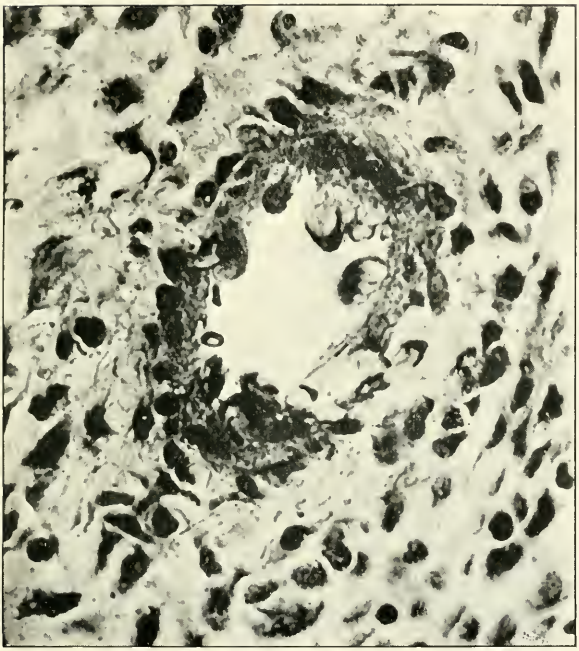
an increase in the intravascular pressure, and a mechanical squeezing of the blood fluid and cells out of the vessels by a process of diapedesis or subsequent to a rupture of the vessel wall.

This explanation has for long been accepted. A careful scrutiny of the microscopic changes reveals its insufficiency. I have already published a preliminary account of this part of my investigations.[1]

In the figures which I show representing vessels in the menstruating mucosa remarkable changes are seen (Plate I.). There is a swelling up of the intimal elements by the accumulation of a clear fluid in their interior, and a displacement of the nuclei and cell substance to the periphery. These changes are always found in a greater or less degree in any condition associated with an escape of the vessel contents, and always in the premenstrual and menstrual states. They are more marked in the vessels which I show first, where there is a thickening of the walls and a forcing into prominence of the changes because of the resistance offered to the fluid-escape. After excluding the possibility of a fatty degeneration or of post-mortem and other irrelevant changes, it is clear that they are associated in some way or other with the fluid-escape. It is seen that there has occurred all along the inner aspect of the vessel wall an active imbibition of the blood fluid, and these appearances, I believe, provide an index of the manner in which there occurs the œdema, which characterises the mucosa in the premenstrual and the menstrual phases. This belief is confirmed by a careful examination of the microscopic changes in the normal mucosa. My specimens are for the most part obtained by curettage, and their preservation is excellent.

In the section of the mucosa, shown in Plate II., the appearances are well shown. We see that the stroma, which

[1] Proceedings of the British Medical Association, 78th Annual Meeting, London, *Brit. Med. Journ.*, 1910, ii., p. 1214.



Menstruation.—Vessel in mucosa with thickened wall, showing marked imbibition by intima. Bulging of cells into lumen. At one place wall teased out by the fluid, and red cell is escaping. Surrounding tissues œdematous.

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under ordinary circumstances consists of fairly well-defined cells united with their neighbours by means of comparatively simple protoplasmic processes, has become completely altered. The nuclei are spread apart, and the protoplasm is beset with a vast multitude of rounded clear spaces of varying sizes, each separated from its neighbours by a complete sheet of protoplasm. The perinuclear portions of protoplasm are equally involved, and the condition is associated with a marked swelling of the originally small cell. The change has led to the breaking up of the protoplasm into fine films, which are sometimes only just recognisable with the highest magnification. At first sight the appearance is somewhat like a fine network formed by interlacing fibrils, and this has hitherto been the interpretation given. We now know, however, in view of the observations I have recorded, that this is an erroneous description. This so-called network, which appears under such conditions, is an index of the functional changes for which the stroma is so perfectly adapted. The fine protoplasmic processes are not filaments, but films, which completely enclose the fluid spaces. I have pointed out elsewhere that the communications between the adjacent cells in the resting state are also complete protoplasmic sheets.[1] They correspond to the films separating the clear spaces in the intimal cells seen in Plates I. and II.

The changes are amenable to one explanation and to one only, that there has been a widespread imbibition of fluid from the blood-vessels in response to some change in the stroma. What is the nature of this change? It has been shown within recent times, chiefly on experimental grounds, that to explain an œdematous escape it is often necessary to recognise the existence of an alteration in the tissue colloids, which enhances their affinity for fluid, and this I believe is what is occurring in the stroma in response to the stimulus of the substance (probably ovarian) which is responsible for the menstrual

[1] See footnote, p. 62.

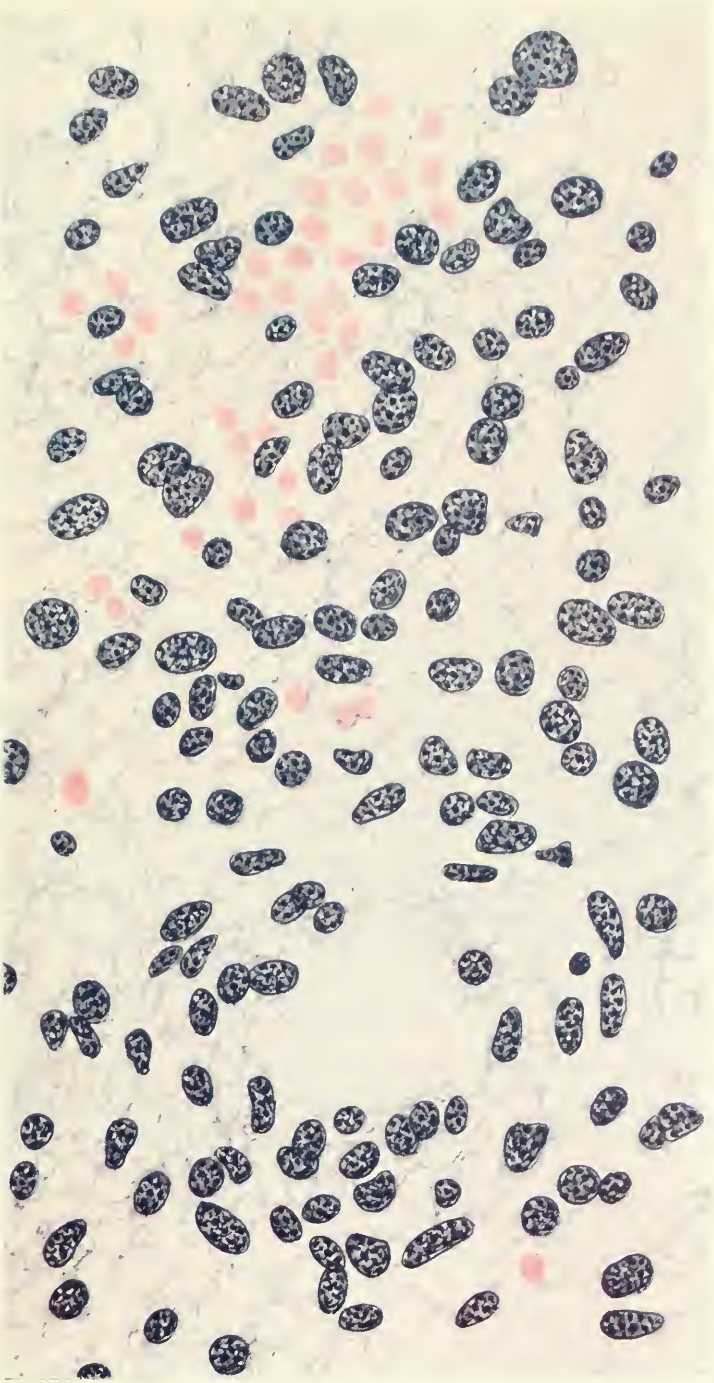
changes. The intimate chemical changes time will not permit me to touch upon.

The changes which I have described enable us to see the stroma in a new light. During these processes any cellular distinctions previously present become levelled and the tissue is seen to consist of a uniform protoplasmic mass. It has for long been recognised that, in connection with menstruation, the uterine mucosa is markedly soft and pulp-like (Möricke, etc.), and the ease with which the displacement of the tissue occurs is clearly dependent on this consistence. The soft, mobile nature of the stroma protoplasm explains at once the manner in which the vessel walls are teased out and the ease with which the surrounding stroma is displaced. That *the stroma consists of a spread-out and undifferentiated connective tissue* is confirmed by the discovery that into its composition there does not enter any specialised form of tissue. Except in the very deepest levels there is a complete absence of elastic, ordinary fibrous and muscular tissue in the stroma and its blood-vessels. These points I have elsewhere referred to, and I have shown that, for some reason, *the vessels, which enter the mucosa from the muscular coat, throw off their supporting coats almost as soon as the stroma is reached.* This peculiar structural feature must have a bearing on the functional changes which occur during menstruation and pregnancy.

How does the blood-escape occur? There can be little doubt that it is determined by the same factors as lead to the fluid-escape. It is easy to understand how, after the fluid opening out of the vessels and stroma the more solid elements escape, and this they do in response to the stroma alterations that tend to drag the fluid into more and more distant levels of the stroma to satisfy the increased affinity for fluid. These observations show that throughout its entire extent the stroma is a *potential blood sponge.*

PLATE II.

Showing structure of stroma of the uterine mucosa and the changes which occur in the premenstrual escape of fluid from the vessels (× 1000). The tissue is opened out by blood fluid, and there is some hæmorrhage. The intercellular spaces are expanded; they are, in most places, enclosed by complete protoplasmic partitions. In addition, the stroma protoplasm is broken up by a multitude of fluid spaces of greatly varying sizes. The spaces are separated from one another by protoplasmic partitions, and their formation has led to a marked swelling-up of the original "cells." The condition is clearly due to a fluid imbibition by the protoplasm, and the partitions are clearly sections of complete films which envelop the imbibed fluid. The change has involved the stroma uniformly, and indicates that this tissue consists of a homogeneous soft protoplasm. It is seen that the so-called network corresponds to the appearance given by a section across the protoplasmic films. In lower part see fine vessel, whose wall is formed by elements structurally identical with the surrounding stroma. Here, again, see evidence of fluid imbibition, which is leading to a teasing-out of the wall. Note the displacement of the protoplasm on the inner aspect, which follows the imbibition—it is by this process that the expansion of the vessel into a "sinus" is taking place.



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Mode of Action of the Chorionic Epithelium.

Before passing on to describe the bearing of these observations, or any attempt to explain the changes in the pregnant mucosa, I wish to refer to some investigations which I have carried out for the purpose of discovering the exact nature of the action of the chorionic cells on the maternal tissues. An understanding of this action, taken in conjunction with the special structural features of the mucosa as revealed in the menstrual changes, will be found to provide a clue to many of the secrets hitherto wrapped in mystery. In the first place it would seem unlikely that a special adaptability, by which at any part of its surface the vessels and stroma can open out, resulting in a flushing of the mucosa with blood, should be overlooked, and, as the orthodox conception demands, that the developing ovum should obtain its blood supply by an entirely different process. The mode of action of the chorionic epithelium is easily studied in the maternal tissues which are the seat of an abnormal fœtal invasion—*e.g.*, the pregnant tube, chorion-epithelioma, etc. In the uterine mucosa during pregnancy, as I shall show, the exact nature of the chorionic action is soon obscured by the decidual change. The same is not true of the abnormal sites of fœtal invasion, where the maternal changes are often clearly traceable to a comparatively late stage of growth.

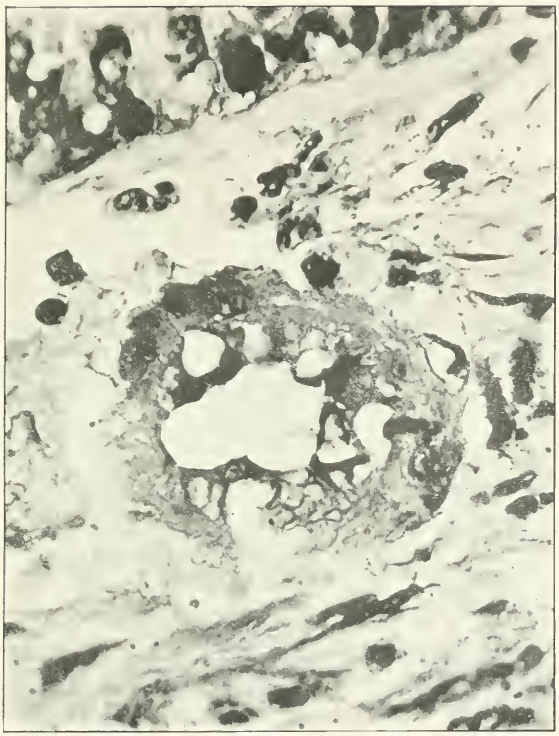
Pregnant Tube.

In the *pregnant tube* there is present a marked degenerative softening. This is most marked in the proximity of the chorionic elements, where the disintegrative process often culminates in a wholesale solution of tissue. But it is also present at a long distance from the actual site of the fœtal cells. One of the most prominent changes, in addition, is a widespread œdematous and hæmorrhagic escape from the

vessels throughout the tubal wall, which again, though most evident near the ovum, is always distinct at a considerable distance.

The orthodox description of the changes implies that the blood-escape, which occurs into the intervillous space, and the more distant œdema and hæmorrhage, are due to different processes. The former—*i.e.*, the immediate cause of the opening-out of the mother's vessels—is, according to the ordinary conception, determined by a direct erosion and destruction of the vessel walls which the chorionic cells encounter in their growth. This allows of a mechanical liberation of the contained blood. That this is not so, and that the blood-escape in this condition is not dependent on two different processes, is suggested by the fact that, as the sections of the tube wall are traced towards the ovum, the blood and fluid leakage, which at the most distant levels are poorly marked, become progressively more and more evident until at the intervillous space there occurs the excessive opening-out of the vessel walls.

What is the cause of the more distant œdema and hæmorrhage? In Plate III. is shown a small vessel at a distance from the chorion. We see that its wall, which has been transformed into a homogeneous degenerating substance, is being teased out by the formation of clear œdematous spaces. These are present in and under the endothelium, which is often bulged towards the lumen. As in the menstrual mucosa, this appearance is dependent on an imbibition of the blood fluid. These changes have at one part led to an almost complete opening-out of the vessel wall. In the next figure this has occurred, and the red cells are seen to be escaping. In this section (Plate II.) changes in the connective-tissue elements similar to those exhibited by the intima—namely, an active fluid imbibition—have occurred. It is easy to show that throughout the entire extent of the tubal wall there are occurring protoplasmic changes which lead to an increase in



Pregnant Tube.—Vessel at considerable distance from ovum, showing mode of opening-out of wall. Note imbibition by intima, the œdematous spaces in the degenerating wall, which at one part is almost completely opened-out. Surrounding tissues œdematous.

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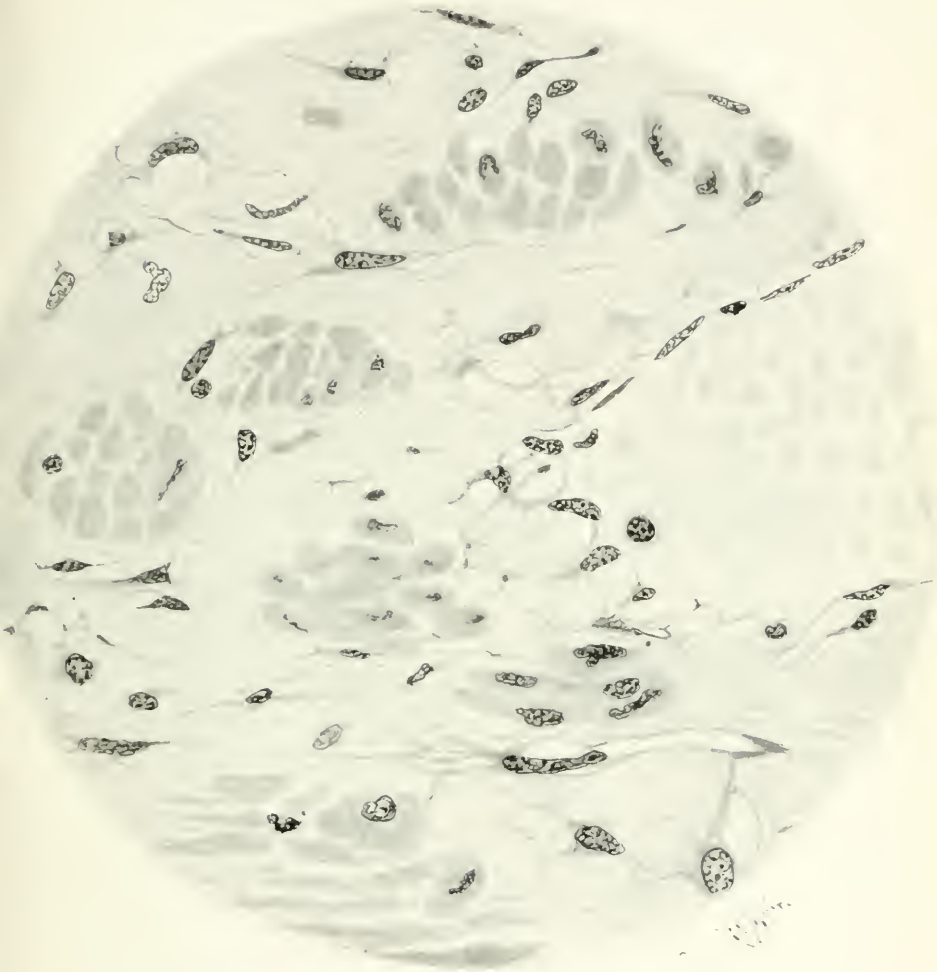

Tubal Pregnancy.—Portion of tubal wall at long distance from chorionic elements, showing changes in intimal and connective-tissue cells and the mode of expansion of the blood sinuses. Note the marked fluid imbibition by the cells, leading to the formation of intracellular fluid spaces, rounded, oval, or elongate on section. To the left side of the sinus the mode of expansion is seen; the displacement or disappearance of the fine protoplasmic films results in a carrying out of the lumen. The mode of formation of the new capillaries is also indicated.

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the affinity of the tissues for fluid. These changes explain the widespread œdema which is dependent on active dragging of the blood fluid into the tissues, and they explain the teasing apart of the vascular walls and the blood-escape. These changes, I believe, explain the gaping of the vessels in the neighbourhood of the ovum and the appearance which is invariably noticed—namely, that the blood cells are streaming across the spaces intervening between the vessels and the intervillous space, and this where there are no cells in the least resembling fœtal cells.

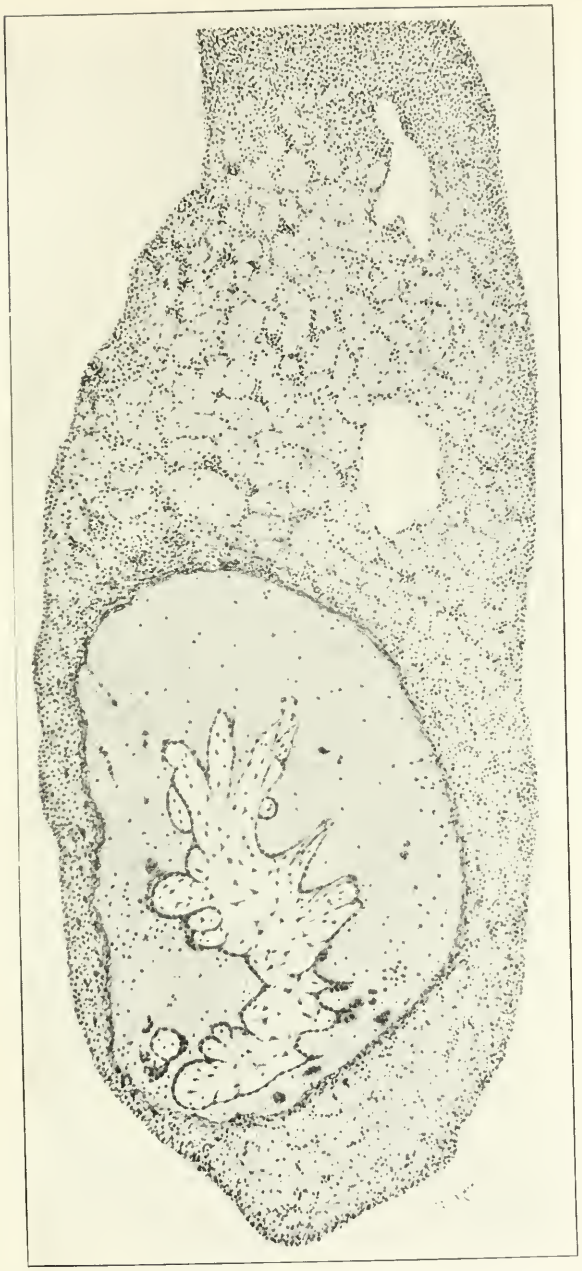
In connection with this I wish just to touch on another point. In the menstrual mucosa it is often easy to demonstrate that *the expansion of the vessels is dependent on exactly the same process as determines the blood-escape;* the opening-out of the wall is due to an imbibition of the fluid followed by a displacement. (Plate II.) In the pregnant tube the same factor, I believe, explains the sinus formation which occurs to such a marked degree. At this part we see the change in action (see Plate IV.). It is interesting to note that in the process of expansion the connective-tissue elements are embraced as a new intima. Here we see a swollen intimal cell in relation to a connective-tissue cell. The disappearance of this fine film carries the lumen a corresponding degree outwards. In Plate IV. also, appearances are shown which suggest that *the new capillary formation which occurs in the pregnant tube is due to exactly the same factors as determine the general fluid- and blood-escape.* Here, however, the blood is being drawn along definite channels formed by an amalgamation of the intracellular fluid spaces, which are well shown. I refer to these changes because of their importance in connection with the pregnant mucosa.

Chorion-epithelioma.

The same changes are revealed by a study of *chorion-epithelioma.* Here, again, there are the same necrotic changes

in the maternal tissues which, though most evident at the point of contact of the chorionic elements, are still present at a long distance. The same is true of the œdema and hæmorrhage. In this plate is shown in the upper part a mass of chorionic cells. At a little distance from this there is a free hæmorrhagic escape from a fine vessel. This is occurring through an apparently intact wall, and certainly before the chorionic bud has reached it. The tissues are œdematous, and in the vessel, in the lower part, is seen the process which, I believe, dominates the mechanism of the fluid- and blood-escape. The wall is swollen up to many times its wonted size, especially on the inner aspect. This is clearly due to a fluid imbibition. In the fine vessels the dragging of the fluid into the surrounding tissues in response to this change soon carries out the blood. This can occur in the thicker vessels only after their walls are teased out. But a careful study of the changes reveals without doubt that a direct infiltration of the chorion is unnecessary. In fact, so far is it from being true to state that the blood can escape only after the vessel walls are destroyed by the fœtal cells, that the converse is the case. The fœtal elements can often be seen growing in the direction of a vessel, from which the contents are already freely leaking. That the muscular elements share in the protoplasmic alterations that enhance the affinity of the colloids for fluid is shown in many places, where there is the same fluid imbibition by the degenerating fibres, which in the process swell to many times their original size. In the case of the muscle, however, there is a speedy breaking-up and solution of the cell substance. This is comparatively rare in the intimal and connective-tissue cells.

A study of the pregnant tube and the chorion-epitheliomatous uterus, then, has shown that there is induced by the presence of the chorionic elements a widespread alteration in the tissues which leads to an active dragging out of the blood fluid and cells from the vessels. It is this change



Early Human Ovum.—Elongated blastocyst lying in cavity in mucosa, surrounded by maternal blood. The surface is studded with rudimentary villi, and is covered with a complete Langhans's layer and, in places, by syncytium. Marginal lamella of decidua necrotic. Mucosa opened out by fluid and blood, even at long distance from ovum. (× 65.)

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PLATE VI.

Early Ovum.—Section through the mucosa in the immediate proximity of the ovum (1000). Above is seen the blood cavity bounded by the necrotic lamina. A degenerating cell (maternal) is seen. Note especially the changes in the stroma protoplasm. The "cells" have become swollen, some in a truly decidual fashion. The preponderating change, however, is seen to be a displacement of the protoplasm by the formation of an immense number of clear spaces of greatly differing sizes. The perinuclear protoplasm is involved in the process which is due to a widespread imbibition of fluid. Into the spaces formed red cells have passed. Between the "cells" there is the so-called network, which is seen to consist in reality of the fine protoplasmic films separating the fluid chambers. Scattered about degenerating stroma cells are seen. Similar changes are present during menstruation. (See Plate II.)

which leads to a flushing of the fœtal surface with the requisite maternal blood. It has for long been recognised that the changes in the pregnant tube, etc., all point to the existence of some biochemical materials or ferments, which are elaborated by the chorionic elements, and which spread into further and further levels of the maternal tissues. All the facts indicate that the changes which I have noted are caused by this secretion. A convincing proof is furnished by the extraordinary degeneration and solution of the maternal tissues which occur at the point of the chorionic contact, and by the fact that these changes spread into the surrounding tissues in ever-widening circles. In none of my sections have I ever seen any evidence of a phagocytic action.

Cause of the Mucosa Changes in Normal Pregnancy.

In the pregnant mucosa the blood-escape is due to exactly the same cause. Plate V. is a low-power section of a very young ovum, which I have recently obtained.[1] We see the irregular blastocyst with rudimentary villi lying in a cavity surrounded by maternal blood. The marginal lamella of the decidua is markedly necrotic. The maternal tissues are infiltrated with fluid and blood, and this at a long distance from the ovum. The coloured drawing (see Plate VI.) is a high-power section of a part of the decidua taken from the neighbourhood of the blood cavity. In it we see the deeply staining necrotic lamina, and we see remarkable changes in the enlarged stroma cells, changes similar to those present in the menstrual mucosa, and the cellular elements of the pregnant tube, etc. We see the fluid imbibition by the stroma elements as evidenced by the appearance of the clear spaces surrounded by the displaced protoplasm, and here,

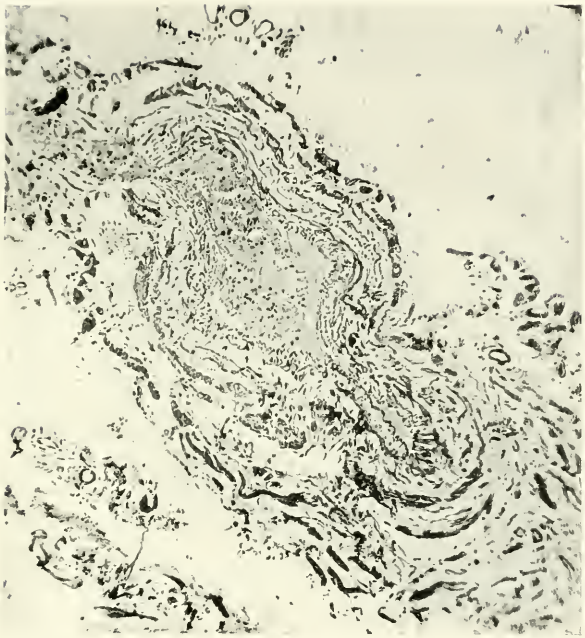
[1] I have published a full description of this ovum, incorporating an expanded account of the menstrual changes and the action of the chorionic cells: *Reproduction in the Human Female*, Messrs Wm. Green & Sons, Edinburgh and London, 1911.

again, we have the appearance of the so-called network, the significance of which I have already noted. The same changes are seen in another young specimen which I have had the opportunity of studying through the kindness of Dr Teacher, of Glasgow. In it there are the intimal and stroma changes, to which I have already referred. In it there is also seen appearances suggesting the formation of new capillary vessels, which I believe occurs in the pregnant mucosa in exactly the same fashion as I have described in the pregnant tube.

It is well known that round the young ovum one of the most remarkable changes in the mucosa is seen in the production of "distended capillaries" or sinuses formed by one layer of cells. These sometimes attain enormous proportions. They are found in all the young human ova, and they are an almost invariable condition in the lower animals. In view of what I have said, it is likely that they are formed, not by a mechanical expansion as the result of increased intravascular pressure, but in response to the tissue changes which lead to the œdema, and which, throughout the stroma, are responsible for the increasing displacement of the soft protoplasm (*cf.* pregnant tube, menstrual mucosa). I believe, also, that the implantation chamber is formed in a similar manner. Space prevents me from entering at length into many of the investigations which are recorded in full in a larger work.

Function of the Decidua.

If the preceding part of this thesis be proven, I have shown that the chorionic cells are concerned in the elaboration of a chemical substance, or substances, which spread into the maternal tissues and lead to the changes which result in the flushing of the fœtal surface with maternal blood fluid and cells. This escapes from the vessels, not mechan-



Pregnant Tube.—Vessel from same level of tube as that represented in Plate VIII., Fig. 1. Note the wholesale œdematous teasing out of the vessel walls. The vessel is almost completely detached from the surrounding tissues.

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ically, but is, as it were, actively drawn through the tissues towards the ovum. This chorionic substance, whilst exerting its most potent influence on the immediate maternal tissues, is continually spreading into more and more remote regions. This is seen in the pregnant tube and the chorion-epitheliomatous uterus, where the maternal tissues are disintegrating and are ploughed up by œdema and blood and by the expanding blood sinuses. These changes are always marked at a considerable distance from the chorionic elements. In this figure (Plate VII.) I show a section of a vessel at a long distance from the ovum—this was situated in the ampulla, whilst the section of the vessel is at the inner end of the tube. We see the œdematous teasing apart of the supporting elements, which has so opened out the wall as to allow of a free hæmorrhagic escape. This is a change which is never found except in pregnancy, and it is amenable to one explanation, and to one only, namely, that the chorionic material has so altered the tissue constitution as to lead to the marked changes present. These slides (Plate VIII., Figs. 1 and 2), which are sections of the pregnant tube at some distance from the chorionic elements, are intended to bring out what I believe is the function of the decidua. We see the marked tearing up of the muscular coat by œdema, and, in places, by hæmorrhage. In the mucosa, the folds of which are turgid by a decidual change in the cells, we note a complete absence of this change. The decidual alteration has prevented it. In this slide we see the close packing of the cells and an absence of a fluid- and blood-escape (Plate IX.).

I believe that these observations provide us with a clue to the function of the uterine decidua.

In the preceding part of this demonstration I have shown:—

(1) That the uterine mucosa is so specially adapted by

the finely adjusted constitution of its stroma as to allow of an immediate opening-out of the vessels and surrounding tissues, and a plentiful flushing of any part of its surface with fluid and blood. This is determined by an alteration in the protoplasm with an increased affinity for water. These changes I have described in menstruation.

(2) That the chorionic epithelium is endowed with the faculty of elaborating a secretion which is intended to act on the maternal tissues in such a way as to lead to these changes.

I have shown that these changes are, in the abnormal regions of chorionic activity, widespread. There is no attempt at a localisation except in the decidually altered mucosa.

A study of the young ovum—*e.g.*, that of Peters, Teacher, and Bryce, and that which I have referred to—reveals the existence of similar maternal changes. There is an excessive opening-out of the vessels and a wholesale displacement of the stroma by fluid and blood, and by the expanding sinuses. Here, again, the changes are evident, even at a considerable distance from the ovum. In the older decidua, on the other hand, these changes, as is well known, are localised to the region where they are necessary.

In view of these considerations, I would venture to assert that we are provided with a definite explanation of the decidua. *Were it not for the alteration in the stroma elements during pregnancy, the uterine mucosa would, in response to the ever-widening spread of the extra-chorionic influence, be torn up by the œdema, blood, and increasing sinuses.* Once the decidual change is definitely established, these changes are localised and inhibited. In view of the special structural feature of the stroma (*cf.* menstruation) we can now understand the reason and the necessity for the decidual change occurring throughout the entire extent of the mucosa from fundus to internal os. In this way only we can account for the *decidua*

FIG. 1.

Pregnant Tube.—Transverse section of inner end of tube, in ampulla of which a two months' pregnancy had developed. Note the œdematous tearing-up of the muscular coats. To the right side and above there is a large track of hæmorrhage. The mucous folds are turgid with decidual cells. This is the only region of the tube where the œdema and hæmorrhage are absent.

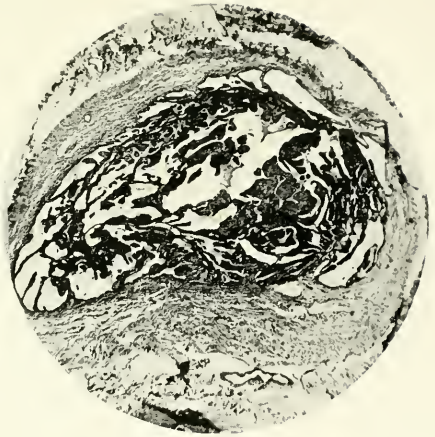
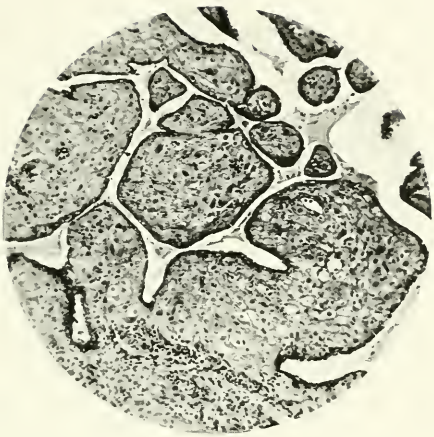


FIG. 2.

Pregnant Tube.—Oblique section through tube on inner side of gestation sac. Note again the opening-out of the muscular coats and the marked œdema round the vessels in the lower part. The mucous rugæ are swollen with decidual cells. Here there is an absence of the œdema, and an appearance resembling the decidua compacta and spongiosa of the uterus. The outer open tissue corresponds to the spongy layer, the inner more densely packed portions of the folds to the compacta. The indentations on the blood-clot present in the lumen suggest that before hardening the closely packed folds corresponded even more to the uterine compacta.



Pregnant Tube.—Mucous folds distended with decidual cells. Note the absence of œdema and hæmorrhage, and the fact that even the fine vessels present are well supported.

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vera. Moreover, it is the existence of maternal blood at any part which allows of a chorionic increase. By preventing the fluid- and blood-escape, the *decidua serotina* and the *decidua reflexa* prevent an excessive increase in the implantation cavity, and an uncontrolled outward growth of the chorionic elements. By this means the chorionic increase is localised to the intervillous space. This interpretation amply accounts for the absence of an excessive invasion by the chorionic elements. Any other action—*e.g.*, a specific antagonism to the chorionic cells—is, in view of what I have said, unnecessary.

For much of the material employed in the investigations I am indebted to Dr Haig Ferguson. For the early ovum I have to thank Dr James Ritchie, Superintendent of the Laboratory of the Royal College of Physicians, Edinburgh, in which a large part of the investigation was carried out.

For the plates, which are shown, I am indebted to Messrs Green and Sons, my publishers, and to the editor of the *Transactions of the Royal Society of Medicine, London*, in which a short account of my work appeared.

The following is a short summary of my work:—

Summary.

(1) In the uterine mucosa and muscular coat during pregnancy there is evidence of a widespread tissue change, tending to lead to an opening-up of the vessels and an irregular œdematous and hæmorrhagic escape. It is present throughout the entire extent of the uterine wall, and it is due to the gradual extension of the chemical substances elaborated by the fœtal cells which constitute the extra-chorionic influence. This extension occurs by a direct passage through the tissues or by a circulation along the blood-stream.

The stroma of the uterine mucosa is especially constructed to permit the occurrence of an immediate blood-escape at any

part of its substance. It consists of a spread-out, soft protoplasm, imperfectly differentiated and richly supplied with vessels, whose walls are composed of the same mobile protoplasmic material. Periodically, there occur the widespread œdema and hæmorrhage, associated with the premenstrual and menstrual phases, in response to some substance reaching it *via* the blood-stream, which alters the protoplasm in such a way as to lead to an "imbibition" of the blood fluid and corpuscles. The same changes occur in the mucosa round, and at a considerable distance from the early ovum, and they are induced near and at a considerable distance from the fœtal elements as the result of the extra-chorionic influence in the pregnant tube and in chorion-epithelioma. This special susceptibility of the mucosa to these changes must be controlled during pregnancy.

(2) In the abnormal regions of chorionic activity, the only sites where the limitation of the vascular and tissue changes has occurred are the places where a decidual change in the connective tissue has taken place. Elsewhere there is a marked œdema and bleeding.

(3) In the mucosa during pregnancy the changes are limited to the region of the ovum (*i.e.*, the place where it is necessary), subsequent to the development of the decidual membrane. This prevents an irregular and increasing opening-up and expansion of the maternal vessels and a tearing-up of the mucosa by fluid and blood after the requisite blood supply is obtained. It is by virtue of this action that the chorionic invasion of the tissues is limited, and that the increase in the implantation chamber is prevented by an erosion or displacement of tissue. These considerations, and these alone, thus amply account for the necessity of a decidual transformation of the entire stroma (*i.e.*, the entire susceptible tissue), and they provide an explanation of the functions of serotina, reflexa, and vera.

The changes in the muscular wall are exhibited in the softening and increasing expansion of the fine vessels which occur.

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The President said he was sure the members of the Society would wish him to thank Dr Young for having brought this demonstration before them. It showed a great amount of work. The subject was of such a technical character that personally he was unable to make any remarks; but the Society would be glad to hear anybody who had anything to say.

Dr B. P. Watson said he would like to congratulate Dr Young on what had evidently been a very large piece of work. It was unfortunate that he had had to run over it hurriedly, and omit many explanations which to some of them were necessary in order to follow exactly the changes described. While he could not accept entirely all Dr Young's interpretations of the appearances shown, he agreed with him regarding the changes in the maternal tissue elements, visible a long way in advance of the chorionic invasion. Mr Wade and he (Dr

Watson) had shown these same changes before the Society some eighteen months before in a case of tubal pregnancy. They then pointed out that these might be produced by some enzyme-like substance. Dr Young had shown them many slides in which the swollen endothelial and connective-tissue cells were seen. Was that imbibition of fluid really an active process? Some of the appearances shown seemed to him to resemble those occurring in dead or dying cells. Was the nucleus in these cells active, and did it stain well? If these cells were degenerating, might it not indicate a preparation for the chorionic advance into the tissue and along the vessels? With regard to the nature of the cells lining the vessel walls of the endometrium, he could not agree with Dr Young that they were connective-tissue cells. He agreed that the cells lining the vessels and those of the stroma were very much alike, but to his (Dr Watson's) mind the more likely explanation was that the stroma cells were endothelial rather than that the cells lining the vessels were connective-tissue cells. If these cells imbibed fluid as Dr Young described, how did they become transformed into the ordinary decidual cells? He was surprised that Dr Young, after so effectively disposing of the mechanical theory of the chorionic advance into the maternal tissue, should fall back on a mechanical theory to explain the power of the decidua in limiting that advance. He thanked Dr Young for his extremely interesting and suggestive paper, the work embodied in which he was sure they would hear still more about.

Dr Haig Ferguson said he was sorry Dr Young had not had time to fully explain the question. One could understand how this opening up took place in connection with pregnancy; but the same change apparently took place in connection with menstruation. What special biochemical substance was present in menstruation? He asked this for his own information, in order to know how to explain it.

Dr Lochhead said he had listened with exceedingly great pleasure and interest to Dr Young's remarks and watched his series of wonderful slides. He would like to draw the attention of the Society to the great importance of the œdema, which occurred in the early stages of pregnancy throughout the mammalia. Not only was the uterine substance swollen and spongy, but a great quantity of fluid was poured out from the cells or between the cells into the uterine lumen. It had a double function to perform; in the first place it acted as a defence to the fertilised ovæ against accident; and, in the second place, it acted as a nutritive. He could not agree with Dr Watson that the process was a degenerative one; he looked on it as a physiological process with the functions he had indicated. In some animals the lymph exuded was not so notable in amount. It had been shown that the underlying spaces were filled by a lymph-like fluid. No explanation had been offered hitherto as to how this occurred; he thought Dr Young had brought that a step forward by referring it to the cells of the stroma. As Dr Haig Ferguson had pointed out, the change also occurred during the constructive period in the menstrual cycle, and it was this conjunction of facts that led one to suspect that the ovary might have something to do with the changes. The ovary was held by some to act as an internal secreting gland. As to the question of an enzyme, Dr Young suggested that an enzyme might be poured into the tissues. He (Dr Lochhead) had not been able to discover this, and until it had been discovered he thought that theory must remain as a supposition.

Dr Fordyce asked how the intervening tissue escaped? Why was not all the surrounding tissue affected in the same way?

Dr Young in reply thanked the Society for the manner in which his paper had been received, and for the flattering remarks of several of the Fellows. He regretted that the short

time at his disposal prevented him from doing more than merely indicate roughly the main lines of his conclusions, which had taken several years to arrive at. It was impossible to give more than the briefest résumé of what had meant several years of work and thought, and those whom he failed to convince, he would refer to a full account of his researches, which would be published shortly. Several workers before (*e.g.*, Wade and Watson) had recognised changes to which he had referred, and which he had described for the first time in the normal pregnant mucosa and in chorion-epithelioma. Nobody before had, he believed, recognised the significance of these changes. In reply to Dr Watson he could only say that there could be no doubt as to the existence of a diffuse protoplasmic change in menstruation and pregnancy, leading as he (Dr Young) had shown to the wholesale fluid- and blood-escape. The error of the ordinary interpretation of the menstrual and pregnant changes was easily demonstrated. The ultimate nature of the protoplasmic alterations he was unable to offer any opinion about. He certainly would be the last to postulate a "vital" explanation, which meant no advance. That it was, on the other hand, not wholly degenerative was proved by the fact that the same fluid imbibition was responsible for the new capillary formation and the expansion of the fine-walled vessels, which occur in menstruation and pregnancy. Neither of these two vascular changes could be considered as degenerative. This was the only length that he was able to go. With regard to the description of the uterine mucosa which his research had led him to formulate, he would remind Dr Watson that it was now universally admitted that the stroma was a poorly differentiated or embryonic *connective* tissue. Leopold's description of thirty years ago, to which Dr Watson seemed to cling, must be given up in the light of succeeding research. He (Dr Young) had demonstrated the exact similarity between

the intima, media, and stroma elements, and he had defined the stroma as a spread-out protoplasmic mass. It was soft and mobile, and the structural features of it enabled the easy opening out of the vessels, and made it really a *potential sponge, and not, as Leopold had described it, merely a lymphatic surface.* As regards the exact nature of the decidual change, he would leave the discussion of this interesting subject to the fuller publication to which he had referred. He was glad to have the support on many matters of such a distinguished worker on this and kindred subjects as Dr Lochhead. He could not agree with Dr Lochhead about the action of the ovaries during pregnancy. The main cause of the immediate mucosa changes must undoubtedly be some material or materials elaborated by the chorionic cells. Everything pointed to it. The changes here, and in the pregnant tube and in chorion-epithelioma, are most marked at the point of the chorionic contact, and fade away more and more as the chorionic elements are left.

Meeting III.—January 11, 1911.

Dr F. W. N. Haultain, *President, in the Chair.*

I. The following gentleman was elected an Ordinary Fellow of the Society:—A. L. S. Tuke, M.B., Ch.B., 12 Comely Park, Dunfermline.

II. *Dr Haig Ferguson* showed—UTERUS WITH SUBMUCOUS FIBROIDS, removed from a single woman, æt. 19 or 20 when first seen. The patient was bleeding very severely; she had a large uterus; and it was thought she might be pregnant. She was curetted twice with little or no result. Subsequently she got married; the symptoms became greatly aggravated, and she was again curetted. Ultimately the uterus was removed and a large number of submucous fibroids were found. There were no fibroids in the uterine muscle itself. The large number of submucous fibroids that might be found was extraordinary, and in a previous specimen, which he had shown to the Society, there were as many as ninety. Dr Fordyce had shown a somewhat similar specimen. It was a very unusual variety of fibroid tumour. In this case one ovary was left; the other, being diseased, was removed. Such a condition in a young woman of 22 or 23 years of age was rare.

III. *Dr J. W. Ballantyne* showed—TWIN FŒTUS ACEPHALUS. The specimen resembled one shown to the Society by Dr Ballantyne fourteen or fifteen years ago, but it differed in so far as it was doubtful whether it had a heart or not. As in all such cases, the fœtus was one of twins. The history was important. At her previous pregnancy the mother had had a male child born at full time; but a few days after its birth sores formed on the hands, and shortly afterwards the child died from exhaustion, the result of hæmophilia. The mother's

father was more or less a congenital imbecile; her paternal grandparents, according to the doctor, "drank like fish"; her paternal uncle was markedly feeble-minded; and her husband was somewhat juvenile—altogether a disastrous sort of heredity. The first twin, a male, a six-months' fœtus, breathed after birth; it seemed normal. The twin which the specimen represented came second. There was one placenta. There was a good deal of liquor amnii. The fœtus was curious, and many years ago, when these cases occurred, and if one referred to the old text-books, one found that the umbilicus had been mistaken for the mouth—a by no means difficult thing to imagine. The lower extremities were clubbed. By means of the X-rays a backbone could be seen running up to somewhere about the middle dorsal region.

IV. NOTE ON A CASE OF LANDRY'S PARALYSIS OCCURRING DURING PREGNANCY AND THE PUERPERIUM.

By Dr Samuel Davidson, Kelso. (Communicated by the Secretary.)

Mrs B., who is now 41 years old, has had eight children (seven of whom are living). She has had no miscarriages. With the exception of her seventh pregnancy, all her pregnancies and confinements have been normal. Towards the end of her seventh pregnancy, that is to say about 15th December 1906, she began to complain of pains in her legs and feet, and had considerable difficulty in walking; she also at this time suffered from troublesome vomiting. She was delivered on 6th January 1907 of a dead-born child. The labour was easy and natural, no instruments being required. Three days afterwards she could not move her legs at all, and still complained of pain in

them. Within the next few days the muscles of the trunk and arms became progressively paralysed. She was quite helpless, unable to feed herself or to turn in bed, or to move either hand or foot, and had wrist-drop and pointing of the toes. There was no paralysis of the muscles of respiration or deglutition. Retention of urine occurred during the first week and required the use of the catheter for four months. With the exception of the pain in the feet there was no disorder of sensation, there were no bedsores, and no electrical changes in the muscles, and not much wasting. In May 1907 improvement first showed itself in the arms, but the finer movements of the fingers were a very long time in being established. The patient suffered from internal strabismus before her illness, but it became worse during her illness and improved as the paralysis passed off. The patient became pregnant in September 1907, and was delivered of a living healthy child in June 1908. She informed me that she thought she would not recover until she had another child, but whether this was an afterthought or not I do not know. Certainly this eighth pregnancy did not make the paralysis any worse though it may have retarded her recovery a little. About September 1908 she began to walk with the aid of two sticks, and she continued to require the aid of a stick in walking until a few months ago. At the present time (June 1910) she suffers no pain, but occasionally feels cramp in her arms and legs. There is apparently only one muscle now remaining paralysed, and that is the extensor proprius hallucis on the right side.

She was well nursed throughout her illness. The only medicinal treatment was the administration of arsenic and strychnine after the acute symptoms had subsided. As regards causation I should be inclined to regard it as one of the toxæmias of pregnancy. The only reference in the literature at my disposal is in Church and Peterson's *Nervous and Mental Diseases*, p. 367, where Dr Church states that among other

causes "pelvic cellulitis, the puerperium, and obscure febrile disturbances have seemed to play a part in its inception." Dr J. W. Ballantyne, to whom I am much indebted for the trouble he has taken, has been able to find only one reference, viz., F. Lewitton, "Landry's Paralysis complicating Pregnancy," from the *North-Western Lancet*, Minneapolis, vol. xxii., p. 318 (1902).

The President thought the Society should thank Dr Davidson for having sent the report of this interesting case. As the members had heard, it was an extremely rare condition; if they had more of these communications on rare cases it would be more satisfactory to them all, and would add materially to the value of the *Transactions*.

Dr Ballantyne agreed with the President that they should thank Dr Davidson, and also that it would be a valuable addition to the *Transactions* to have these rare cases published. Before Dr Davidson realised it was such a rare condition he had consulted Dr Ballantyne, who looked up the literature and was only able to find one definite reference under Landry's paralysis.

V. ACHONDROPLASIA, BRACHYDACTYLY AND CRANIO-CLEIDO-DYSOSTOSIS: THEIR NATURE AND CAUSATION—A PRELIMINARY SKETCH.

By D. Berry Hart, M.D., etc., Lecturer on Midwifery and Diseases of Women, School of the Royal Colleges, Edinburgh, etc.

Certain congenital deformities in the human race are distinguished by the fact that in them the deficiency is very exact; further, they occur apparently suddenly, and are transmitted sometimes in exact ratios. We have thus an exceedingly

interesting problem to solve, of which hitherto no solution has been advanced.

I shall first describe three of these deformities and then consider an explanation of them. I wish to point out, however, that the usual term, deformity is one based on an æsthetic point of view. They are best considered, I believe, from the point of view of "variation," and it is in this light that I take them up for special reasons to be given presently.

I now go on to describe and explain these conditions, and I have selected Achondroplasia, Brachydactyly, and Cranio-Cleido-Dysostosis as the chief examples.

Description of the Deformities.

Achondroplasia.

In this interesting condition we have an individual with short legs, short arms, deficient length of the ribs, and deficient ossification and length of the basioccipital. This is, therefore, a deficiency in quite a definite group of bones, and it has been shown by Symington, Alexis Thomson, and John Thomson, that the bones affected in this variation are those which arise by early endochondral ossification, and the deficiency, I urge, is one in their unit-character of length.

I now describe an exceedingly rare and striking condition, namely:

Hereditary Cranio-cleido-dysostosis.

In this there is at first deficient ossification of the bones of the body developed in membrane, viz.: the vault of the cranium and part of the clavicle.[1] This has been carefully considered recently by Mr D. C. L. Fitzwilliams.[2] There are minor

[1] See Fawcett's Long Fox lecture, "The Development of the Human Skull," *Bristol Med. Chir. Trans.*, June 1910 (vol. xxviii., p. 108), for a full discussion of this question; also, *Quain's Anatomy*, 11th edition, "Embryology," by T. H. Bryce.

[2] *Lancet*, Nov. 10, 1910, Fitzwilliams.

degrees of this where the whole clavicle, that part developed in membrane and that in cartilage, is awanting, or where the two halves have not united.

In the complete or fairly complete cases of this condition, a large part of the cranium remains membranous during infancy, with very large fontanelles and an open frontal suture (Metopy). Ultimately, ossification takes place about the twentieth year, and gives a skull with a curious bossed arrangement in its vault. The face and the base of the skull are only secondarily affected by being opened up, and the want of complete ossification on the outer part of the clavicle, leads to its appearance in some cases in two parts.

Brachydactyly.

In this condition the hands are short and stumpy, and the middle phalanges are represented, as Drinkwater has shown, by a nodule, the basal epiphysis of the second phalanx, ankylosed to the terminal phalanx. There are also some height-variations in Brachydactyly, which I do not now consider. Drinkwater has also shown that in a certain number of cases in families where they can be traced, the ratio is 50 per cent. normal hands and feet, and 50 per cent. abnormal (Brachydactylous). Farabee's cases were initiated in America by an English immigrant with this peculiarity of the hands and feet.

It will be noticed in the conditions of achondroplasia, brachydactyly, and cranio-cleido-dysostosis I have described that we have (1) some deficiency in an exact group of tissues marked out definitely in their development; (2) that so far as we know, the individual starting this condition did so suddenly, that is to say, in the form of what is termed in ordinary phraseology, a sport, or better still, a mutation, according to de Vries. This variation, when it occurred was (3) transmitted in certain cases, in definite ratios, and was transmitted unaltered.

This is of very great importance, because the Darwinian view of small variations accumulating in their repeated transmission, so that in crossings with contrasted characters, blending and improvement takes place, is not borne out at all in the instances I have given. Mutation once started is transmitted in an exactly defined manner. How then are we to explain all this?

I have first to consider some points in the mechanism of evolution. The crucial point in any mechanism of evolution is—How does a variation occur, how is it transmitted, in what ratios do the unit-characters segregate, and are they unaltered or gradually transformed?

We are at present all agreed that evolution is at work in vital processes, but we have to do more than that. We have to give the mechanism by which evolution occurs. On this, as yet, our knowledge is of an elementary nature.

Darwin took it for granted, or held at any rate, that variations occurred, and he seriously grappled with the question of their transmission. The opinion evidently held by him, and the one that is now generally adhered to, is that a variation occurs in the soma or body of the plant or animal, and in order to be causal, has such an effect on the germinal portion or propagative portion that it puts it in the position of transmitting it. Now it is a great thing to recognise that, for transmission, the germ cell must, in the case of a new variation, have or acquire something that transmits the variation.

Darwin formulated a theory which did not satisfy himself, namely, that when a variation occurred, say for instance that due to injury, gemmules were sent from it to the sexual glands; these became causal and transmitted the variation to the posterity of the plant or animal. This theory has been quite abandoned, but the search for such a relation between the soma and the propagative part goes on as eagerly as ever.

Semon, a distinguished naturalist, has evolved the elaborate theory termed Mnemism. He supposes that when a cell is altered by what produces the variation, an engram or record is formed in it, and then by nerves or protoplastic strands, an impression is conveyed to the germ cells, and this impression, becoming causal, transmits the variation.

It may be said of all theories of such relation between the altered soma and the germ cell, that they are wild in the extreme, and yet passionately held by many of their originators. The actual evidence of the transmission to posterity of an injury is of the most insignificant description, and therefore it should have been really a great relief to biologists when Weismann boldly said that acquired peculiarities could not be transmitted. This view of Weismann is I believe the correct one; but few may agree with me when I say that it gave the *coup de grâce* to all attempts to make a somatic injury, or an alleged variation due to environment or diet, etc., causal in the germ cell of the plant or animal somatically so affected. This position, viz., that a somatic variation may be transmitted, however, is tenaciously held, because at present there is no other means available of explaining the fact that variations occur and are transmitted, and this really makes the dilemma of evolutionists.[1]

In considering this question there are some preliminaries to be noted. In the first place we must look at the terminology. We are far too apt to speak of abnormal bodily conditions in the human race as "deformities." It is a more dispassionate term if we use the word variation. Thus the achondroplasic man is to a certain extent deformed, but that is rather an æsthetic view of him. He is really a short-legged variety of mankind, usually quite intelligent, and we have nothing to do

[1] In present Darwinian circles it is not recognised that extrinsic variation is not the only possibility. There may be intrinsic variations, as we shall see.

with any other consideration at present. A similar deformity has been found to occur in sheep, the well-known case of the Ancon sheep, for instance, and in the Dachshund.

A second point arises in regard to variations, namely, that variations are constantly occurring although some of them are not capable of being transmitted. For instance, we have anencephalic fœtuses occurring fairly frequently in human pregnancies. These, however, do not survive, and therefore a race of them cannot be established by heredity. In the same way we may look upon malformation of the genital organs. We may have non-descent of the testes, which almost always involves sterility, or there may be some hypospadias which will tend in the same direction. Then again various atresias of the genital tract turn up with a fair amount of regularity in women, although these atresias necessarily make the women sterile. I might multiply many instances of such, but the point to be emphasised is this, that variations are constantly turning up in a certain number of cases and in a proportion we do not know as yet, although the possessors of them are in virtue of this variation sterile. The first thing one thinks of at present in any variation is—What about its heredity? In the instances given above there can be none. The non-transmission of acquired peculiarities, and the want of a mechanism to explain how environment, if it makes a variation, can have this transmitted from soma to germ cell, leads me to start the inquiry—May variation not arise from causes intrinsic in the primitive germ cells and zygote?[1] That it does so I now try to show.

If we examine biometrically those who make up a large school, or a regiment for instance, and if we make in each of

[1] By a *primitive germ cell* I mean the structures in the ovary or testis, usually termed germ or sperm cells, after their karyokinetic changes; by a gamete, a matured primitive germ cell, the ordinary ovum or spermatozoon which has matured by the reduction of its gametes to one half: by a zygote, a fertilised ovum.

these cases certain measurements of stature, weight, circumference of chest, length of any special part of the body such as fingers, toes, and so on, and plot out our results, arranging them in regard to the numbers for each length ascertained, it will be found that the curve plotted out gives some form what is termed the Curve of Probability.

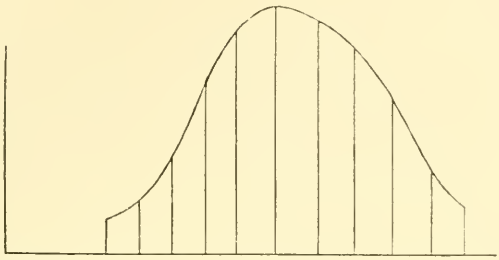


FIG. 1.—FREQUENCY POLYGON, showing the distribution of measurements of an "organ" in a sufficient sample of the population (Worthington).

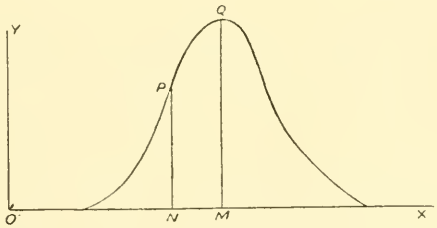


FIG. 2.—The same as above, but with a more continuous curve, owing to a greater number of observations (Worthington).

This curve of probability,[1] or some of its modifications, seems indeed to guide our growth and all the functions we perform.

In plants, for instance, if a sufficient number of pea-pods are measured so as to get what is called a typical pod *qua* this special measurement, it will be found that the measurements

[1] The subject of biometry was started by Quetelet, and is now the line of inquiry employed by Galton, Pearson, and many others.

vary; but if we take a sufficient number and plot out the numbers, putting the extremes of length and shortness, necessarily few, at each end, and grading the others between, we shall find again that this will give us once more the curve of probability. The same may be done in regard to leaves, and in regard to anything which is the result of vital development. It holds also good for questions of birth-rate, death-rate, incidence of crime, legitimate births and illegitimate births, and so on, so that this curve of probability is really in one sense the curve of normal and abnormal vital processes or of life. An immense number of observations has been accumulated and is still being accumulated as to its value by statisticians, and this is of the very highest importance. We must ask, however—What is its meaning? Why should this curve govern the bodily and functional processes of mankind?

For some years past embryologists have been working on an obscure and difficult part of their subject, one that is by no means popular either with students or biologists, namely, the very earliest processes of fertilisation and also the earliest stages of the development of the sexual glands. Thanks to the labours of a large number of observers we have now attained in regard to these processes an amount of knowledge which, however, has not been correlated, and I now go on to consider these facts, and bring forward an attempt to correlate them.

(1) *Early Development of the Sexual Glands.*

Here at a certain stage, when the embryo is formed, the germ epithelium of the genital ridge of the Wolffian body, a thickened layer, which is continuous with the ordinary peritoneal epithelium, has in it what are known as the primitive sex cells or primitive germ or sperm cells, according as they are found in the ovary or testis. Waldeyer, to whom we owe the discovery of this fact, gave the only interpretation then possible, namely, that the primitive germ cells were transformed

germ epithelial cells. Two things have shaken this view—(*a*) the discovery that during development, in early developing elasmobranchii, before the embryo is formed, the germ cells are found in the blastoderm and yolk stalk *en route* apparently for the sexual gland, and that they have been derived from an early division of the zygote when it had the determinants of heredity in it in a causal condition. From what we now know of Weismann's and Mendel's work in regard to the nature of the germ plasma and the unit-characters,[1] and from the view now taken that determinants[2] of the special unit-characters for all the developing organs and functions of the body are present in the chromosomes of the nucleus of the zygote, and also, of course, in those of the gametes, we see the absolute futility of considering complicated structures like the primitive germ cells (primitive ova) as being derived from ordinary somatic cells like those of the germ epithelium. The most rational view of the germ epithelium is that it is a protective epithelium protecting the germ cells until they are embedded in the substance of the ovary, and even then acting as protectors for them in the shape of the cells of the membrane granulosa. The theory, therefore, as we may modestly term it, that the primitive germ cells are derived from an early division of the zygote, that at the earliest period of zygotic development we thus get the primitive germ cell mass formed, and that from the zygote the primitive germ cells arise, as well as the somatic portion giving ultimately the individual plant or animal, gives us a view of the matter that is not only I believe correct in the main, but most suggestive and helpful in making us really understand what heredity is. We see at once that the gametes and the zygote contain the unit-characters of heredity because

[1] The term unit-character is a Mendelian one. It expresses the idea that the individual is made of structures and qualities which are autonomous.

[2] Determinant is Weismann's term for the protoplasmic agents in the chromosomes of the germ cells and zygote causal to the unit-characters.

they are derived from the zygote at the period when the latter has these in such a condition that they are causal and capable of infinite multiplication. (*b*) After these primitive germ cells get into the germ epithelium they become embedded in the substance of the ovary probably by a mutual action, the connective tissue of the ovary growing out and snaring them in as Foulis described, and the primitive sex cells making their way into the substance of the ovary perhaps by their own movement. It has been noted by Beard that the primitive sex cells while in the developing organism, but not in the sexual glands of the elasmobranch, do not undergo mitosis, and this seems to me of great importance. When, however, the primitive germ cells are in the substance of the ovary they undergo mitosis, bringing about the changes (prematuration) of the ovum, specially described by von Winiwarter, Miss Lane-Claypon Bryce, Miss M'Ilroy, and others. The view that most observers take of this mitosis is that it exhibits the phases of transition between the germ epithelium and the primitive germ cell. The germ epithelium, therefore, according to this view, is a most remarkable structure, because not only are the primitive germ cells derived from its somatic cells, but a certain number of the germ epithelial cells with such powers, afterwards become the mere protective covering of the ovary and the membrane granulosa cells of the Graafian follicle. The view that I take of this process will be developed in a little after I have considered Mendelism, but I may say in anticipation that I consider the mitosis as being a means of causing variation in the determinants of the unit-characters lying in the chromosomes.

We have now to consider:

(2) *The Earliest Changes that take place Prior to Fertilisation.*

The primitive germ and primitive sperm cells contain in them the chromosomes, and these contain the determinants of

the unit-characters of heredity. In the prematuration changes of the ovum or sperm cell these undergo mitosis, *i.e.*, variation as we have seen, and now we have to record a very remarkable phenomenon seen best in white mice and in the *Ascaris megalocephala*, namely, that prior to fertilisation the approaching gametes lose half of their chromosomes in the form of the well-known polar bodies. By this means—maturation—they are now reduced primitive germ cells, and have only half the number of chromosomes of the ordinary somatic cell or primitive sex cell. By their union the zygote is formed, and thus the number of chromosomes made up to the normal standard.

It would take too long time to discuss all the views that have been given as to the significance of the polar bodies. The one that I advance at present is that the throwing off of the polar bodies is a second means of variation of the unit-characters. In the prematuration changes of the ovum and sperm cell, variation has taken place, and here we have prior to fertilisation another effective means of producing a variation zygote, as half of the chromosomes are thrown off and the half that remain are theoretically either invariably the same or they differ in each case. The latter seems to me a more probable supposition, as we shall see presently. The polar bodies thrown off may contain ordinary determinants, or those whose loss produces a marked variation or mutation, and that is of importance.

We have now to go on to the question of what takes place in—

(3) *The Early Condition of the Zygote.*

I have suggested that the determinants in the chromosomes are varied in the prematuration changes of the ovum. A second variation arises in the fertilisation stage, and now we have to ask—What happens to the determinants when they are blended in the newly formed zygote? We can describe this accurately now, thanks to the researches of Mendel.

Mendel's work is now well known and I have in previous papers described it. I therefore give only the general description, and the one necessary for the subject I am now considering. Mendel, in such a crossing as the well-known one between a tall and a dwarf plant, showed that in the first generation of the hybrids, as he termed them, or F^1 as we now say, the character of tallness was present in the plant and that of dwarfness not expressed in it. I may say that Mendel and almost all the Mendelians who follow him, except myself, take, in the schemes of Mendelian crossing, the *plant* and the *animal* only, *quâ* the segregation of the contrasted qualities, and we shall see presently that this involves them in a very serious difficulty.

There are really two schemes of Mendel's results, one actual, Diagram 1, and the other diagrammatised, Diagram 2.

Mendel therefore found that the tallness was present in the first generation of plants, but the dwarfness was not expressed. He called the tallness the dominant quality and the dwarfness the recessive quality. Now when this generation is allowed to self-fertilise we get a second generation, F^2, in which tall plants and dwarf plants are present in the proportion of 3:1. This is expressed in Diagram 1, which is the accurate diagram in Mendelism. The reading of it is, therefore, that the parent crossing gave rise to the F^1 generation, all tall, and that F^1 on self-fertilisation gave tall plants and dwarf plants 3:1; the dwarfs bred true subsequently; the tall plants gave on self-fertilisation one-third which bred true to tallness, and two-thirds which bred again 3:1 of tall plants to dwarf plants. It is to be noted that the tall plants in F^1 differ as to their future, the tall plants in F^1 always giving one-third which breed true to tallness, and two-thirds which do not breed true. Now this is a little complicated and difficult to grasp until one has thought it over, and accordingly, Diagram 2 gives a much more easily grasped formula, for here we see in the first

generation that the talls and impure talls and dwarfs are segregated out in the special ratio of 1:2:1, which may be termed the Mendelian ratio. This second diagram, although easier to grasp, is a misleading one, as we shall see presently.

Diagram 1.

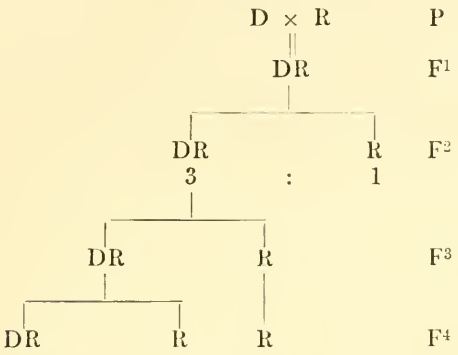
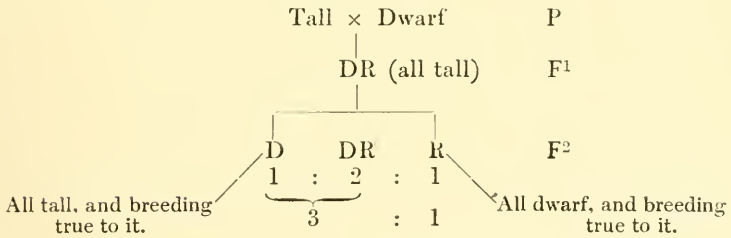


Diagram 2.



Diag. 1 and 2.—D= Dominant, the tallness; R= Recessive, the dwarfness; and DR= impure dominant, *i.e.*, a plant which, although tall, produced pure Talls and impure Talls, 3:1. The tall character Mendel termed Dominant; the dwarf, Recessive. Pure talls and pure dwarfs give only talls and dwarfs respectively. × = crossed by or fertilised by.

In F^2, D:DR:R as 1:2:1, the Mendelian ratio apparently *in the plants* (*vide*, however, p. 94).

Mendel's facts have been tested in the most extensive manner, and have sustained the test most satisfactorily. We must remember that Mendel's results took more than one season to bring out, at any rate in the case of contrasted

characters in plants, and when he got his results out either as to plants or peas, and especially when he obtained the Mendelian ratio of 1 : 2 : 1, he saw that it was a probability result, that is to say (if A represented the dominant quality and B the recessive quality), $(A+B)^2$ gave $A^2+2AB+B^2$, *i.e.* A : 2A : B, 1 : 2 : 1, the squares being neglected as not affecting a quality. We thus see that the 1 : 2 : 1 ratio was obtained.

To explain this, Mendel supposed that the pollen grains and egg cells (or the microspore and macrospore) contained the quality of tallness or of dwarfness as the case might be, pure in one of them.[1] Purity of the gametes, therefore, *quâ* the unit-characters, or as it is termed, gametic segregation, was the view brought forward by Mendel, and he supposed that this being the case the Law of Probability gave the Mendelian ratio.

Mendel therefore did the greatest service in showing that the qualities and functions of the plant and animal can be considered as unit-characters, that these are autonomous and do not blend in their combinations, and segregate out in a certain ratio—the Mendelian ratio of 1 : 2 : 1, by what has been termed the Law of Segregation.

Now, as already said, I consider that there is an error in this Mendelian theory, and it is an error of the very highest importance. It may be put in this way—If the Mendelian ratio in which the tallness and dwarfness segregate out, is due to a combination of gametes which are pure, relative to contrasted characters, we ask at once why this ratio does not come out in the plants of the first generation. It really only comes out in the plants when we take the results of the second generation and the third generation, and then when we plot these out in the second diagram we seem to get out a ratio 1 : 2 : 1 in the second generation. This, however, cannot be the

[1] For convenience and to bring animal and botanical embryologists into the same terminology, we may speak of the microspore and macrospore as gametes.

case, as the combinations of the microspore and macrospore must end in a generation, and should not, on this theory, take two generations before we see the results in the plants.

A further point in regard to this is that those who hold Mendel's theory of gametic purity and combination are in a difficulty when you ask them a very simple question, namely, In the first generation of plants where is the recessive quality of dwarfness? We are told that it is in the soma of the plant. The question then is—If so, how is it expressed? To that they give really no answer. It must be in the plant somewhere or other, as it comes out in the second generation. Now it is remarkable to note what Mendel answered to this question: "The expression recessive has been chosen because the characters thereby designated withdraw or entirely disappear in the hybrids, but nevertheless reappear unchanged in their progeny, as will be demonstrated later on." (Bateson's translation of Mendel's paper in Mendel's *Principles of Heredity*, Cambridge, 1907, p. 325.)

It is most characteristic of the remarkable intellect of such a man as Mendel that, in stating his views on this difficult point as to what had come over the recessive quality in the first generation, he uses the remarkable word "withdraw." He had evidently what may be termed the Clerk-Maxwell intellect, namely, one of those that is incapable of thinking wrongly on biological or physical questions. One has only to complete it by saying, "'Withdraws' to the propagative part of the plant, and thus is expressed in the soma of a succeeding generation plant."

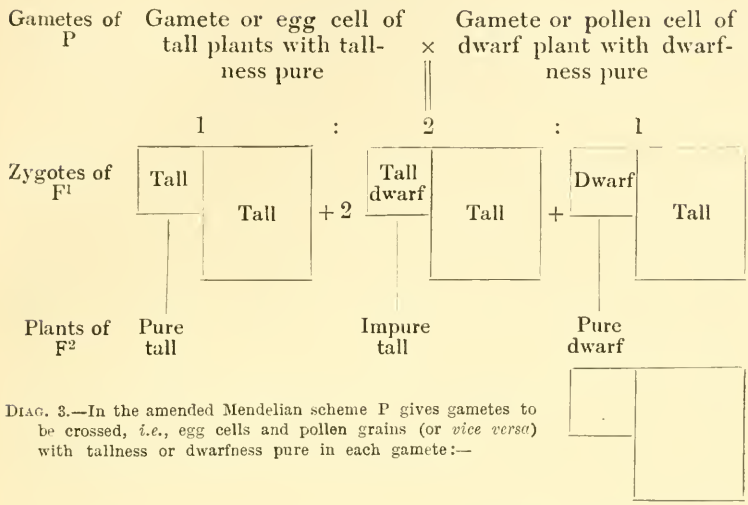
The solution I propose is that the plant be considered as being made up of a propagative part and a somatic part, and that when the zygote is formed, a propagative and somatic part is developed, so that we get a condition analogous to what is

found in the animals higher in the life scale. In botanical embryology the matter as to the early origin of what will form the pollen grains and egg cells is not as yet clearly worked out, but there are facts that point to something like an analogous condition. For instance, the microspore and macrospore form three cells in their maturation, one of which is the generative cell and the other the vegetative cell, the last forming the pollen tube for instance. Then, again, the oospore, which is the equivalent of the zygote, is divided into two parts which give definite parts of the plant, termed the upper or epibasal and a lower or hypobasal. From the former are produced the young stem, first leaf, and the cotyledons, whilst from the lower are the foot and the so-called hypocotyledonary (H. A. Haig, *The Plant Cell*, Griffin & Co., 1910).

If, then, we have this division of the plant zygote into a propagative part and a somatic part, we can arrange the Mendelian diagram as to transmission of qualities in a very simple and clear manner. Thus, when a tall and dwarf crossing is made, the microspore and macrospore, the one containing the dwarfness pure and the other containing the tallness pure, meet and form a zygote. It must be noticed now that I am making this scheme of Mendelian transmission *one of zygotes and then of plants.* In the first generation, then, we really get as the result of the cross-fertilisation three types of zygote. Each has a propagative part and a somatic part, and as in the first generation all the plants are tall the somatic part of the zygote must, therefore, contain the tall determinants pure. As a quarter will breed pure tall, the propagative part will contain the determinants for tallness pure. As a quarter breed true to dwarfness, the propagative part of this quarter will have dwarf determinants; finally, as half of them breed tall plants and dwarf plants 3:1, we have in this propagative part both the tall and

dwarf determinants. The following scheme shows this diagrammatically:—

Diagram 3.



Diag. 3.—In the amended Mendelian scheme P gives gametes to be crossed, *i.e.*, egg cells and pollen grains (or *vice versa*) with tallness or dwarfness pure in each gamete:—

gives figure of a zygote, with propagative part the small square, and somatic part the large square; F^1 shows the zygotes on this view, and indicates that the Mendelian ratio 1 : 2 : 1 *is in the unit-characters of the propagative part and not in the somatic part of the zygote.* Note that the somatic part of the three kinds of F^1 zygotes will give only tall plants in F^1, but that the propagative part of the F^1 zygotes will give plants in F^2 and F^3 with the Mendelian ratio.

The reading is this—The gametes in the T × D crossing give three kinds of zygotes in F^1. Inasmuch as the somatic part of all the zygotes contains tall determinants, the resulting *plants* are tall in F^1: as the tall and dwarf determinants are distributed in the propagative part of the zygote as 1 : 2 : 1, the plants in succeeding generations come out in this ratio. All the talls are somatic talls, and all the dwarfs somatic dwarfs. The difference in the talls and *impure talls* is expressed only in the propagative part.

It will be seen, therefore, that the Mendelian ratio really does come out in the first generation, but only in the propagative part, and consequently the Mendelian ratio is really a ratio of the *segregation of the determinants for the unit-characters in*

the propagative part of the zygote. In the somatic part of all the zygotes in the first generation the determinants for tallness are pure. Thus when a zygote is formed the determinants of the unit-characters that are contrasted are really distributed between the propagative part and somatic part of the zygote in such a way that the tallness or dominant determinants are pure in the somatic part of the zygote, whereas in the propagative part they are distributed according to the Law of Probability of error, that is to say, in the Mendelian ratio of 1:2:1. Why the quality of tallness or the dominant one should be pure in the somatic part in the first generation I am unable fully to explain, but it is so. In this way we get a very important point, namely, that the Law of Segregation by which the contrasted characters in the crossing are sifted out in the Mendelian ratio is due to *a combination by probability of the determinants for the unit-characters in the zygote*, i.e., *zygotic segregation, and not to a combination of the microspores and macrospores, as Mendel held.*

Zygotic segregation was suggested to me by an investigation into the nature of the sterile animal known as the Freemartin. I found that it was not a sterile cow but a sterile bull. The freemartin is, therefore, a sterile bull usually born co-twin with a potent bull. On examination of specimens, Hunter's and others, it has been found that the sexual gland in the freemartin is a testis, and I found by studying the plates of Spiegelberg's specimens as well as those of Hunter and one or two examples in my own possession, that in addition to the defective and undescended testes the animal possessed the epoophoron and prostatic utricle of its potent co-twin in whole or part (the non-potent parts of the genital tract). The freemartin and its co-twin are identical twins, that is, derived from one zygote. The essence of the freemartin is practically that the determinants of the non-potent organs have been segregated into the zygote of the non-potent animal. Here, then, we have

this condition of segregation arising in the zygote, and thus applying this to Mendel's ordinary crossing, I found that a complete and satisfactory rendering of the reading of the scheme could be obtained by dividing the plant into a somatic and a propagative part, *i.e.*, by a theory of zygotic segregation as opposed to the theory of gametic segregation. This is embodied in Diagram 3.

We may, therefore, say in general that when changes are taking place in the chromosomes, say of a primitive germ cell, as in the prematuration and maturation of the ovum, or in the zygote as in the Mendelian process, *we have a rearrangement of the protoplasmic determinants of the unit-characters according to the law of probability.*

This view of segregation gives us a simple definition of Dominance and Recession. A quality is dominant when it appears pure in the somatic part of the zygotes of the first generation. A quality is recessive when it appears in the propagative part of the first generation zygotes and not in the somatic. These qualities are therefore better termed the first quality and the second quality relative to their appearance in the somatic part of the zygote. An impure dominant is a plant whose zygote has the determinants both for the contrasted qualities in its propagative part. This simplifies the matter very much, and takes away the idea as to the terms dominant and recessive possessing some mysterious quality which can be invoked in the difficulties of heredity.

A very remarkable point in regard to variation has now to be restated, namely, that the results of variation, in the human race for instance, when taken in sufficient number and plotted out in a curve according to the frequency of the special variations, give the curve of probability. Thus the height of 1000 soldiers, the chest circumference of 1000 soldiers, the length of pea-pods, the length of leaves, the lengths of a phalanx of the finger, when taken in sufficient

number give us this curve, and we may therefore state in a fairly broad way that if the anatomical and functional variations in man are observed in sufficient numbers (for instance, if it is a case of height measurements), and the numbers of the obtained heights all plotted out, we get this curve. The reason of this is, I urge, that in the changes in the determinants of the unit-characters which take place in the prematuration and maturation of the ovum and in the Mendelian segregation of unit-characters, the law of probability is the governing one in determining their segregations, and *thus we get all adult variations necessarily following the same law as that which determines the rearrangement of their determinants in the ovum and zygote.*

This makes variation an intrinsic phenomenon. It happens during the mitosis of the primitive germ and sperm cells in the ovary and testis, and when the ovum matures and the chromosomes are thrown off (polar bodies), and also when the zygote undergoes segregation changes (Mendelism). All these are done by the law of probability, and in this way an enormous amount of variation can be produced.

I do not discuss the ordinary explanations of the cause of variation. The most accepted one is that it is due to Natural Selection, this acting in the struggle for existence in which of course environment is a factor. The struggle for existence seems to me to be an eliminative process, and as such to be of importance. Unfit variations are eliminated by it, but the struggle for existence has nothing to do with causing variations. Environment is urged as a cause by many able observers; but the environment must act, if it does, either directly on the primitive germ cells themselves, or on them through the soma. How environment can act directly on the primitive germ cells seems to me impossible to explain or even to imagine, and that it acts on the germ cells through the soma is excluded by the fact that we now know that

acquired peculiarities are not transmitted, that is to say, the soma has no influence on the sexual gland other than a nutritional one.

The theory that I have advanced will seem of course a materialistic one. All variation is due under it to intrinsic changes in the primitive germ cells, gametes, and zygote, and is thus a process beyond the direct interference of our environment or of the soma of any individual. Whether such a view is materialistic is not the question. The question is—Is it right? It is evident that a great many calculable variations will be produced in this way, and a certain number of them will be unfit for development. They will, however, form a very small proportion of the cases, and their production is an incident of the process and involved in its nature.

The argument so far may be summed up now as follows:—The Mendelian ratio of 1:2:1 is a result of the law of probability, but is not due to a combination of the gametes as such under the law of probability, but to a rearrangement in the zygote of the determinants, and under the same law. I extend this, and suppose that when we have the determinants segregating by this law in the prematuration and maturation changes in the ovary, and also in the Mendelian changes in the zygote, there is a rearrangement according to the law of probability of the determinants, and all this will give "variation" by an intrinsic Mechanism.

There are thus three Mechanisms that may be used for investigation in Evolution problems.

(1) Strict Mendelism; what we may term the First Mechanism.

(2) Mendelism modified by the rejection of the gametic segregation theory and the substitution of a theory of zygotic distribution of the unit-character determinants—the Second Mechanism.

(3) Modified Mendelism, or the Third Mechanism, *i.e.*,

Strict Mendelism with zygotic segregation of determinants instead of their gametic combination, with the view added of the zygotic rearrangement according to the curve of frequency.

(1) *Strict Mendelism.*

Gametic segregation and purity of the gametes to contrasted unit-characters which are so widely accepted, I consider the essential errors of Strict Mendelism. (*Vide ante*, p. 96.) It is of interest to note that Strict Mendelism has not advanced markedly in the solution of any of the difficulties of evolution, from the stage at which Mendel left it, and this despite the brilliant work which has been done by many Mendelians. What hampers Strict Mendelism in investigation is the theory of gametic segregation, as evidently the application of a theory which is wrong cannot make for progress. It also limits the *locus* of probability combination to the union of the gametes. If, however, we take

(2) *Mendelism with the Theory of Segregation*

of the unit-characters in the zygote, according to the law of frequency—the Second Method—we really get on. One can then extend the *locus* of variation, as where we have determinants in the chromosomes of the primitive germ cells and gametes, there a frequency-variation must occur. We must add, however, to this modified Mendelism, the conception of the zygotic origin of the primitive germ cells and that of the gross loss of unit-characters in the polar bodies thrown off when maturation takes place, the

Third Mechanism.

I wish now to apply this third mechanism to the solution of the three special variations I have described.

Achondroplasia.

Here the unit-character determining the length or size of the group of unit-characters, whose ossification takes place early in cartilage, has been thrown off in part in the polar bodies. The result of this is to give a zygote with these determinants wanting, and thus the ultimate somatic part of the individual will be achondroplasic. Thus we have an exact loss and an exact variation or so-called deformity. The zygote giving rise to this achondroplasic would derive its primitive germ cells and gametes from an early division of the zygote, and thus so many of them would lack these special determinants for length. In this way it would be transmitted in certain ratios, but these are not yet known by observation so far as I know. The unfortunate male achondroplasic is not a very eligible suitor, and the women are apt to have pelvic deformity.

Brachydactyly.

In this the variation or mutation is due to the loss of the determinants of the unit-characters for length or size of the middle phalanges of the fingers and toes. The 50 per cent. of these abnormalities I explain by supposing that all the determinant material for this was thrown off. The amount of determinant guiding the size of a single series of phalanges in the hands and feet must be inconceivably small, and might thus be lost in one minute chromomere or minute particle of a chromosome. In these cases, when marriage takes place with a non-deformed person, the results would be like the tossing of coins for head or tail, 50 per cent. of each. It would be either deformity in this respect or non-deformity. Drinkwater supposes that the 50 per cent. is due to one of the parties in this respect being an impure dominant and the other a recessive, this crossing giving as we know 50 per cent. of each. It does not seem to me to

apply here. Here, therefore, modified Mendelism—the second method—gives us the autonomy of the unit-characters and the frequency result; but we need the polar body loss of the third method and the zygotic origin of the primitive germ cells to clear up the whole matter, viz., exact loss, transmission, and ratio. The genesis of this deformity therefore seems to me to have been this. The first individual with the deformity lost *inter alia* the determinants of the unit-characters for length in the polar bodies thrown off at the conception that gave rise to him; he would therefore be born with the middle phalanges deficient and apparently wanting. The zygote that gave rise to him also gave rise to the primitive germ-cell mass; this of course ultimately formed the gametes, and consequently he would transmit it in a certain proportion of cases, 50 per cent. as already explained.

A remarkable illustration of the views advanced is the third and very rare deformity known as hereditary cranio-cleido-dysostosis. This we need only briefly indicate.

Hereditary Cranio-cleido-dysostosis.

From the point of view of Mendelism we must look on the bones which persist in membrane (vault of skull and half of clavicle) in this deformity, as already described (p. 84), as a combined group of unit-characters, which has lost in the polar bodies the determinants of the early ossification of the bones laid down in membrane. Sometimes the whole power for the group is lost and sometimes only part of it, and in the latter case we may have only clavicle defects.

Mendelian Unit-Characters in the Human Body.

From the point of view of ossification, the human skeleton may be thus looked on as split up into three great Mendelian

or autonomous divisions or groups of unit-characters. (*a*) The group developing in early endochondral ossification (limbs upper and lower, ribs and basioccipital); (*b*) the group developing in late endochondral ossification (spine, carpus and tarsus, pelvis, sternum); (*c*) the group developing in membrane (vault of skull, clavicle).

Each of these three great divisions is a group of unit-characters, and has its causal zygotic determinants, structures which have distributed among them what determines the size, shape, ossification, and so on in the developing adult tissues. When the former determining factors are thrown off in the polar bodies we get a variation condition arising in the three deformities already considered, and this is transmitted by the zygotic origin of the primitive germ cells. The ratios are due to the law of frequency governing the determinants in the primitive germ cells.

Variation in General.

It follows from what I have said that variation in general is an intrinsic phenomenon occurring in the primitive germ cells and zygotes, and arising from the changes of the determinants of the unit-characters following the curve of frequency, and that such variations are transmitted by the primitive germ cells being derived from the zygote itself. This makes variation and its transmission an automatic phenomenon governed by the law of probability. The elimination of variation-results is by Natural Selection, but Natural Selection does not create variations.

Pangenism, Mnemism, and even one of the most recent theories which we may term Hormonism, fail to explain the transmission of variation. The reason may, following Weismann, be put thus in general terms. A variation that arises from an extrinsic cause cannot become causal in a reproductive sense. The somatic results of the supposed

extrinsic cause cannot become causal by being placed in the propagative part of the organism, as that which is formed is not formative in a reproductive sense. The somatic results of the supposed extrinsic variation-influence cannot themselves become causal for the future, by appearing or being transmitted to the germ cells, unless we hold that the somatic and propagative parts are equivalent in an advanced organism. Even if we admit that extrinsic variation could be made causal in the sex gland, there is no known mechanism by which this can be effected.

Looked at generally, then, variation in organisms may be considered as an intrinsic phenomenon occurring in the primitive germ cells when these undergo mitoses in the sexual gland, and in maturation when the polar bodies are thrown off. Variations in the determinants are constantly occurring as part of the normal fertilisation and prefertilisation phases, and as the law of frequency governs them we get normally important unit-characters varied and then some of them thrown off in the polar bodies. This is why certain deformities, *i.e.*, variations such as Anencephaly, genital malformation are always occurring although necessarily not transmissible.

I may sum up then as follows:—

1. Strict Mendelism comprises autonomy of the unit-characters, their non-blending, their sifting out in the subsequent generations in a definite ratio, the results giving a frequency-curve.

2. The theory of gametic segregation of the determinants of unit-characters and of the purity of each of the contrasted ones in a gamete, is erroneous.

3. The view of the origin of the primitive ova and sperm cells from the germ and sperm epithelium respectively, mere somatic cells, is unsatisfactory and in my opinion erroneous.

4. The primitive germ and sperm cells arise from an early division of the zygote and thus contain heredity determinants.

5. The mitoses they undergo in the sexual gland bring about variation of the determinants of the unit-characters of heredity in the chromosomes, and in this variation follow the curve of frequency.

6. The fact that measurements of functions and anatomical organs in the adult follow some curve of frequency is due to the view expressed in 5.

7. The polar bodies thrown off prior to fertilisation give a marked and sometimes important loss of determinants leading to variations which may be called deformities, but really give rise to a variety.

8. Variation is an intrinsic automatic phenomenon due to changes in the primitive germ cells (mitosis and maturation) and in the zygote (Mendelism).

9. Human deformities are really variations and may be regarded as mutations.

Dr Ballantyne said it was a very difficult problem to know how to discuss this paper, for Dr Hart had given out a lot of matter some of which he himself must admit was debatable. He would, therefore, confine himself to one or two points of which he had a little knowledge. For instance, with regard to anencephalus, as far back as 1891 he (Dr Ballantyne) wrote a paper on the arrangement of the bones of the head, which Sir William Turner published in his *Journal of Anatomy* at that time. In that paper he tried to point out that it was the bones developed in membrane that were absent; where bones were developed partly in membrane and partly in cartilage, it was the former part that was wanting. In composite bones, like the temporal, the results did not agree with the accepted embryology, but it was possible that the embryologists were sometimes in error in locating the spheres of membrane and cartilage in bone development.

When one reached the other points of the paper it was very difficult to express an opinion; and yet Dr Hart no doubt wanted something more to be said than that one could not follow his paper. To take, again, the question of anencephalus in the case of a woman who gave birth to seven anencephalic infants, it was very difficult to maintain that such a fact supported the Mendelian theory. And the same difficulty occurred with regard to other deformities; for instance, how could Mendelism explain a woman giving birth to malformed children by her first husband and normal children by her second? He was not aware until Dr Hart stated it that mitosis did not occur in the germ cells when they were travelling through the body.

As regards Dr Hart's theory of a "brick out" to explain malformations, he could not see how such a view sufficed in malformations by excess; in them it was rather a brick added than one left out.

He thought Dr Hart had rather overloaded his paper, although the different points had a certain bearing upon each other, and it was a little difficult to give a reasonable criticism of it; at the same time he would not like to omit to say that personally he had listened to it with great pleasure and interest.

Dr Barbour said he had nothing to add in the way of criticism. He admired the enormous amount of work Dr Hart had put into his paper. The paper raised a great number of points. He would ask where the germ cells came from?

Dr Hart said they were derived from the zygote.

Dr Barbour asked what part of the body?

Dr Hart said there was no body at the time, it was just the developing embryo.

Dr Fordyce said he had followed the paper with great interest. He asked if achondroplasia was hereditary.

Dr Hart said it was, but that it had not been worked up. Brachydactyly was better known as to transmission.

Dr Fordyce said the point was that if all deformities were due to the loss of unit-characters, what determined the unit-character and what did not? Did all deformities follow the same law? Dr Hart had taken conditions where there was heredity to prove his point.

Dr Hart said it might not be hereditary at all.

Dr Fordyce said it seemed a most remarkable thing that one of the phalangeal bones should be a unit-character of the body.

Dr J. W. Simpson said if one went on the theory of chance should one not get more achondroplasia?

Dr Hart said he had looked up achondroplasia. Bateson did not describe it.

Dr Simpson said it occurred frequently in the same family, that being so, one would expect to see achondroplasia oftener than one did.

Dr Hart pointed out that there was natural selection.

Dr Simpson said there was a danger for women; the men were absolutely normal.

Dr Hart said the men were sometimes imbecile, or at any-rate mentally defective.

Dr Simpson said they were not imbecile but absolutely normal; John Thomson, who had worked up the subject, he thought more than anyone else, said so.

Dr Hart said he had met with a number of cases and had found mental deficiency in some.

Dr Hart, continuing, said he had written on the subject both popularly and scientifically, but it was a difficult one to make clear. With regard to the questions put, he did not hold with Mendel's explanation of everything, only the autonomy of unit-characters and the ratios. One must keep in mind in addition the fact that the segregation happens in the zygote, *i.e.*, it is in the fertilised ovum that the rearrangement takes place of determinants. As a result of losses in the polar bodies we may

get brachydactyly and cranio-cleido-dysostosis; and it also explained why members of the same family were different from one another, and in the same way we might have a woman with one Fallopian tube, the other tube being lost prior to fertilisation. Distinct variation was constantly going on. Dr Ballantyne was in error in stating that the explanation given was in essence a Mendelian phenomenon. It was based on a loss in the polar bodies. For its transmission, Mendelian autonomy and the early zygotic origin of the germ-cells was required. In regard to Dr J. Ritchie's remark on acromegaly, this deformity was not a congenital one, and therefore did not come into the scope of the paper. The bones involved in acromegaly were developed in cartilage.

Dr Ritchie said Dr Hart's papers were always worth listening to. This one showed a tremendous amount of hard work and a very great amount of originality. If he referred only to the body characters and the reasons of the variations in different members of one family, he (Dr Ritchie) would have nothing to say, but when he made his theory relate to deformities, he had very considerable difficulty in understanding him. Dr Hart had taken up the question of achondroplasia, a condition in which certain abnormalities were found in the long bones, that is to say, in the bones which were developed from cartilage. Now, there was another disease in which the defect related to bones developed in membranes, viz., acromegaly. It was now known that acromegaly was determined by a defect of the pituitary body; what determined the particular defect in achondroplasia was not yet known. It might probably be explained by a defect of some other structural body which affected the bones developed from cartilage.

Meeting IV.—March 8, 1911.

Dr F. W. N. Haultain, *President, in the Chair.*

I. The following gentleman was elected an Ordinary Fellow of the Society:—John Bruce Ronaldson, M.R.C.S., L.R.C.P., Charing Cross Hospital, London.

II. *The President* showed—(*a*) FIBROMYOMA OF VAGINA which was growing from the posterior wall. It bulged very freely through the vaginal wall and gave rise to a considerable amount of pain, particularly on locomotion. The specimen had shrunk very much since removal; he showed it as one of those rare tumours which are met with occasionally in the vagina. So far as the rectum and other surrounding parts were concerned, the tumour was very freely removed, but a considerable part of the vaginal wall had to be removed before it was got away. There was nothing special about the specimen except its rarity.

(*b*) ENCEPHALOID CANCER OF OVARY—BILATERAL. This specimen, from its encephaloid nature, was somewhat more interesting than usual. The progress, as one would expect, was extremely rapid, the patient only having noticed the condition for some two or three weeks. On opening the abdomen one saw the whole of the peritoneum was studded. Some four weeks after the removal of the tumours the patient was extremely well. (He had intended to bring some microscopical specimens to show the encephaloid nature of the tumour more minutely, but had not been able to lay his hands on them.)

(*c*) FIBROCYSTIC UTERINE TUMOUR, WITH TUBERCULAR PERITONITIS, removed from a patient æt. 50, and four years past the menopause. She suffered from extremely severe pain which had only lasted about a month. On opening the

abdomen this tumour of the pelvis was found, matting the two tubes together. The peritoneum was studded throughout with a number of sago-like granules which were typical of tubercle. A caseous condition of the fibres of the uterus was found, and one hoped to find some tubercle; but, as was to be expected, none was found, such a condition was practically unknown. The caseous appearance was very suggestive, however. The tumour was interesting because of the caseous appearance and its distribution, and also because of the age of the patient.

(*d*) EXTRA-UTERINE GESTATION AT FIVE AND A HALF MONTHS. —The President asked if he might be allowed to show this specimen, which had occurred only a short time previously, and there had not been time to get it on the billet. The condition occurred in a woman, æt. 44, who had last menstruated on the 24th of August. There had been no irregular hæmorrhage—complete amenorrhœa. Her youngest child was 14 years of age. She naturally thought herself pregnant again, and that was confirmed by the condition of the breasts and other signs. There was absolutely no pain. On 1st February she suffered from retention of urine with a considerable amount of pain in the abdomen, and there was a little hæmorrhage. The pain was more characteristic of labour than anything else—coming and going. She was seen by her doctor, who gave her morphia or something of that kind. He (Dr Haultain) examined the patient a month later, and detected a mass in the pelvis on the left side, while on the right side of the abdomen there was another mass reaching up to within a couple of finger-breadths of the umbilicus. The two masses seemed to be connected with each other and were of a doughy consistence. In the absence of irregular hæmorrhages and everything else, the conclusion come to was that the condition was a fibromyoma coexisting with pregnancy. A slight attempt was made to push the mass up from behind, but as the cervix was well to the front it did not seem possible that a fœtus could be expelled in this way,

and operation was thought advisable. On opening the abdomen he was very much surprised to find that the mass was the right tube containing the placenta, and it was attached to the other mass which was the ovum in an adventitious sac; the two were attached by the umbilical cord. He imagined that the condition had been an extra-uterine gestation, which had probably been situated at the fimbriated extremity of the tube; it had grown down from that into the abdomen, and had given no signs of rupture whatever. Although this was only a supposition, yet he thought the signs led one to believe that such would be found to be the case. The placenta was still intertubal and had not ruptured the tube. There had been no hæmorrhage into the abdomen. The specimen was interesting on account of the clinical history. Although it was difficult to shell out, it had not given rise to any untoward effects at present; at all events the patient was still alive.

In answer to questions from various Fellows, the President said that the uterus seemed somewhat enlarged. He might also say that the condition could easily have been mistaken for a retroverted gravid uterus, and the other mass a myoma on the anterior wall.

Dr J. W. Ballantyne said he thought the specimen should have something more said about it than just shown as a specimen, and illustrations of it would also be important. He did not remember having met with a record just similar. It was dangerous to say it was unique, but he had never heard of anything like it before.

The President said he had not opened the specimen for he had thought it best to keep it for microscopical purposes. He would certainly make some further investigations.

Dr James Young asked if the child had been long dead.

The President said he did not think so. The mother had menstruated last on 24th August, and the specimen was removed on Saturday last (4th March); symptoms of what he

took to be a spurious labour occurred on the Saturday before that. The specimen looked like a five-months' fœtus. The sac was purely adventitious.

III. *Dr B. P. Watson* showed — UTERUS REMOVED BY SUPRAVAGINAL HYSTERECTOMY FOR INTRACTABLE UTERINE HÆMORRHAGE, removed from a iii.-para, æt. 35. The patient's last child was born three or four years previously; there had been no miscarriages. There had been menorrhagia lasting for about ten days at a time, and the patient lost a considerable amount of blood. Curettage was performed but did no good; the hæmorrhage began again and she became very anæmic. The abdomen was opened and the uterus removed by supravaginal hysterectomy. The wall was found to be very thick; on microscopical examination there was a considerable increase of the fibrous tissue to be seen.

IV. AN EXPERIMENTAL STUDY OF THE ANAPHYLACTIC THEORY OF THE TOXÆMIA OF PREGNANCY.

By R. W. JOHNSTONE, M.D., F.R.C.S.E., Assistant to the Professor of Midwifery, University of Edinburgh.

THE term Anaphylaxis was coined in 1902 by Richet, and, as the word suggests, it was applied to a condition which is the very antithesis of immunity. Much experimental work has enabled the facts to be formulated as follows. Certain organic substances, all of them complex proteins of unknown chemical formula, when introduced for the first time into an organism in sub-toxic or non-toxic doses, have the property of producing after a certain fixed incubation period an excessive sensitiveness to subsequent very small doses of the same substance.

For example, suppose a rabbit be injected with, say, 5 c.c. of,

say, horse serum. After an interval of ten days that rabbit is in a state of supersensitiveness to even a very small second dose of the same serum. The symptoms produced come on almost at once—a blanching of the ears, dyspnœa, palpitation, loss of muscular tone, and in severe cases convulsions, paralysis, and death—all within a very short time.

It is not my purpose, nor am I competent to discuss the cause of this very extraordinary condition. It is believed to be due to the development of certain properties in the blood serum, because this supersensitiveness can be transferred to another animal of the same species by a transference of serum from a supersensitised animal.

It is presumed that the protein first injected acts as an antigen, and induces the formation in the blood of an antibody, to which the name of "anaphylactic reaction body" is given. This antibody requires a certain fixed period of time for its elaboration. At the time of the second injection this anaphylactic reaction body unites with the antigen in the second injection, the union probably taking place in the nerve cells, and so producing the shock. So far as is known, the reaction is a specific one, at least within the same limits as immunity is specific.

Although the subject has as yet been only very faintly elucidated, enough is known to enable us to class an anaphylactic phenomena in the human body such disasters as occasionally follow the administration of horse serum, *e.g.*, antidiphtheric, and antitetanic, and so forth. It explains the local phenomena sometimes seen at the site of injection as well as the general rashes sometimes observed. It is also probably the basic principle underlying the tuberculin reactions of von Pirquet and Calmette, and of the mallein reaction in glanders.

Anderson and Rosenau (1) in 1906, were the first to throw out the suggestion of a possible connection between anaphylaxis and the toxæmia of pregnancy. It is quite true that the points

of resemblance between the two conditions are not numerous. There is clinically the shock and intoxication of the nervous system as manifested by convulsions; and post-mortem the characteristically frequent occurrence of multiple hæmorrhages.

In spite, however, of the apparent lack of similarity between the two conditions, the urgency of the problem of eclampsia is such that every conceivable clue, however slender, is worth following up. It was with this idea that my observations were made, and also because more general observations on anaphylaxis were being made in the Royal College of Physicians' Laboratory at the time. It is a pleasure to acknowledge the kind help and guidance given me by Dr Ritchie and Dr Scott, who were working at the subject on larger and more general lines.

The idea underlying this theory was that some alien protein might pass from the fœtus or placenta into the maternal circulation, and supersensitise the mother: and that a subsequent transference of the same substance after a suitable interval might account for the onset of convulsions and other symptoms. All attempts to produce anaphylaxis with fœtal blood have been negative, and in any case these are of comparatively little interest, as we know that the source of the poison of eclampsia cannot be the fœtus. Anderson and Rosenau (1) succeeded in obtaining anaphylaxis in guinea-pigs with guinea-pig placental extracts, but they allowed the placentas to autolyse for three hours at 37 C. before making the extract. Murray (2), in a recent paper in the *Journal of Obstetrics and Gynæcology*, refers to this point, and records some experiments which he performed with autolysed liver, and which he regards as indicating that Anderson and Rosenau's results were due, not to placental toxins, but to products of autolysis. This, however, was admitted as possible by these observers in their original paper. Still, the point has to be borne in mind, that placenta very readily undergoes autolysis,

and it becomes of more interest still in the light of Dryfuss's work, which proved that the eclamptic placenta undergoes autolysis—due to the excessive activity of its ferments—even during its intra-uterine life.

My own experiments were confined to the use of the placenta only. They fall into five groups.

In the first group I used an extract of placenta from an eclamptic patient. The placenta was obtained within an hour of birth, minced, washed as free from blood as possible, rubbed up with silver sand, and filtered through a sterilised filter. Two female rabbits received intravenously, doses of 3 and 3½ c.c., respectively. Eleven days later they received intravenous injections of 1 and 3 c.c., respectively, of the same juice—the juice having been kept in the interval, at a temperature just above freezing-point. No symptoms were observed on the second injection beyond slight dyspnœa and uneasiness which passed off within a few minutes.

In the second group I carried out similar experiments in twelve female rabbits with the juice of normal human healthy placenta. The fresh placentas were minced, washed free of blood, and the juice extracted by hydraulic pressure. The doses employed varied from 3 to 5 c.c.—in most cases 4 c.c. The interval between the first and second doses were varied from twelve days to twenty-three days. The usual incubation period in the rabbit is about ten days, but it was thought possible that for placental proteins it might be different. In any case there was little fear of missing the supersensitive state, as once it is established in the rabbit it has been shown to persist for at least ten days (3).

In 7 of these 12 cases, slight symptoms were observed—dyspnœa, blanching of the ears, loss of muscular tone, and slight fall of temperature. In an 8th case, more marked symptoms followed the second injection, there being slight convulsions and profuse liquid diarrhœa in addition to the other symptoms.

In the third group the conditions were made as realistic as possible, *pregnant* animals being injected with the juice of placenta from another of the *same species*. In this way I made observations on two rabbits and one guinea-pig, the placenta in each case being removed from a newly killed animal, minced, washed free of blood, and the juice extracted by rubbing up with sand and filtering. In the guinea-pig there were no symptoms after the second injection. One rabbit showed only slight dyspnœa. The other rabbit showed no symptoms. In the interval between the first and second doses, however, it gave birth to three young ones. These were dead when found, and on examination showed no obvious morbid appearances. It has been shown in other cases (4) that the young of mothers in an anaphylactic state are themselves supersensitive to the original protein. Unfortunately the death of the young ones precluded the possibility of testing this.

In the next two groups of observations I used powdered placenta suspended in normal saline solution. It will be recalled that Liepmann believed that the eclamptic toxin is in the cells of the placenta and may not be found in the juice. Liepmann's methods were open to many objections, and a great deal of doubt has been cast upon his observations accordingly. But, whether right or wrong, his work was suggestive, and commanded a great deal of interest, and it seemed worth while to make one or two experiments in anaphylaxis along the same lines. Eclamptic placentæ being unavailable, normal ones were used.

The placentæ were minced and washed as before: then dried *in vacuo* and powdered to a consistence of soft sugar. The powder was a pinkish brown in colour. One gramme of this was suspended in about 6 c.c. normal saline solution and injected into the peritoneal cavity. Owing to the cellular nature of the mixture, intravenous injection could not, of course, be used, and, therefore, one could not expect such a marked anaphylactic shock.

Of four rabbits thus injected with human placenta powder, two died within twenty-four hours, and post-mortem showed congestion of liver and kidneys, and some recent peritonitis. The other two animals survived without any discomfort and received second doses, eleven and twelve days afterwards. There was not the slightest appearance of any anaphylactic symptoms during the twelve hours subsequent to the second injections.

This may be due to the fact observed by Liepmann that the poisonous element is very labile, and that powders kept for any length of time may lose their toxicity.

Lastly—as a fifth group—a similar experiment was carried out on a female rabbit with powder of rabbit's placenta, with the production of only very slight panting and uneasiness—which might merely be due to the injection of a gramme of solid matter into the peritoneal cavity.

To summarise: Anaphylactic symptoms were obtained by the use of normal human placenta juice in eight out of twelve rabbits. This indicates that human placenta juice contains some complex protein body which is capable of acting as a toxin for other animals.

No results followed the use of eclamptic placenta juice. So far as it goes, this may be taken to lend support to the view that the toxic element had passed out of the placenta into the patient's body.

No results followed the use of extracts of animals of the same species. I failed to confirm Rosenau and Anderson's results, but the placentas that I used were absolutely fresh and there was no likelihood of autolysis having occurred before use.

Finally, no results followed the use of powder of placenta, either human, or of animals of the same species.

The question remains—Were such anaphylactic symptoms as were obtained, due to a placental toxin, or to the small quantity of blood serum inevitably present? It is impossible

to wash all the blood out of a placenta by any method yet devised—either the simple one employed in these experiments, viz., washing the minced placenta in a muslin bag under running water, or the more elaborate, but not more effectual one, of injecting water into the umbilical vein immediately after birth.

At first sight one is inclined to put down the positive results to the contained blood serum—and for the following reason. All experiments in which placenta of the same species was used —that is to say, all experiments in which any contained blood serum was *not* an alien protein—were negative. These were the cases in which one could disregard entirely the presence of the homologous blood serum, and had they shown any results, these results would have been undoubtedly due to placental alien proteins. These cases, however, showed no anaphylactic symptoms.

Against this must be placed the fact that Scott (3) found as a result of much observation on rabbits, that the minimum dose of human blood serum capable of producing anaphylaxis in the rabbit was about 1 c.c. I used doses of 4 c.c. of placenta juice, but I am quite confident that blood serum was not present in it in anything approaching such a quantity as 25 per cent. I believe, therefore, that the positive results were due to placental elements. I am inclined also to think that these elements were not products of autolysis, as the placentas used were perfectly fresh.

Several observers have noticed that the effects of placental extracts are not uniform. This I also found to be the case. The three most marked results that I got in the second group were all produced by the juice of one particular placenta, which appeared to the naked eye to be healthy and normal. It was treated in the same way as the others, and contained, so far as I could ascertain, neither more nor less blood than the others.

The results of these experiments, particularly the failure to

confirm Anderson and Rosenau's results with homologous placental extract, appear to support the view that there is no evidence of anything of the nature of anaphylaxis, so far as we at present understand that subject, in the commoner manifestations of the toxæmia of pregnancy.

References.

1. Anderson and Rosenau. — *U.S. Marine Hosp. Service, Hygiene Lab.;* 1906, Bulletin 29, and 1807, Bulletin 36.
2. Murray.—*Jour. Obst. and Gyn. Brit. Emp.*, 1910, xviii., p. 225.
3. Scott.—*Jour. Pathol. and Bacteriol.*, 1910, xiv., p. 147.
4. Lewis.—*Jour. Exper. Med.*, 1908, x., p. 11.

The President said there was no doubt the Society had to thank Dr Johnstone most heartily for his paper. Although the results had been negative they showed an amount of work which was both instructive and valuable. There could be no doubt that the subject of eclampsia at the present time was one of the main questions to be solved; it seemed to be the curse, if he might say so, of obstetrics, particularly in Scotland; and in his opinion any work which was done with a view to solving the mystery, or throwing light which would enable them to treat the condition on scientific lines, could only be most heartily encouraged. They were indebted to Dr Johnstone for making these experiments, and he hoped he would continue his work on this important subject. It was important for the Society that the younger Fellows should take up some subject and work it out.

Dr A. H. F. Barbour said he did not know sufficient about the subject to criticise or offer any suggestions, but he would like to emphasise what the President had said as to the value to the Society of young men taking up subjects such as this

and working them out. Although Dr Johnstone's observations had been negative, it was a splendid piece of work.

Dr J. W. Ballantyne said he thought it was a pity the word anaphylaxis had come in at all; as Dr Johnstone had almost hinted, it really meant nothing, indeed in the Greek it had no meaning. He thought the word "supersensitisation" a better one, as it gave a better idea, really meaning supersensitiveness. He had been very much interested to hear from Dr Johnstone some little time ago that the results were negative, because one was almost led to that conclusion *a priori*. It did not seem a sensible suggestion that there was an anaphylactic element in it. He quite agreed with the President and Dr Barbour, that although an experiment was negative nevertheless it was experimentally instructive, and negatives should always be brought forward. He recalled one negative result of Dr Helme's, namely, tapping the spinal canal. There was a great wave of feeling in its favour at the time. He (Dr Ballantyne) had tried it, and published a case, but the patient died; he gave it a second trial, but had never tried it again. That method of treatment had passed out of account altogether. If a negative result is not published, the original observation is apt to gain weight and really do harm, particularly in other parts of the world where it gets quoted. He did not think it probable that the fœtus or the placenta supplied the toxin. In his experience he did not find that not emptying the uterus interfered with the result.

Dr Oliphant Nicholson said he had listened to Dr Johnstone's paper with very great interest. Although the experiments seemed to show that eclamptic symptoms were not due to a supersensitiveness on the part of the organism (anaphylaxis) to any substance which could be extracted from the dead placenta, still he believed that everything pointed to this organ being intimately concerned in some manner with the origin of eclampsia. The condition was due to a toxin circulating in the

blood-stream. Where the poison came from was, as yet, unknown, but clinically the most striking evidence of its presence was a great rise in the blood-pressure due to intense and widespread vaso-constriction. In impending eclampsia these circulatory features could not fail to attract attention. So the toxin of eclampsia acted like adrenalin or pituitary extract on the circulation. The placenta must be regarded as a gland with an internal secretion, the functions of which were not yet established, and when Dixon and Taylor, in 1907, asserted that human placental juice contained a substance which contracted the arterioles and raised the blood-pressure even more strikingly than adrenalin, one began to think that the placental secretion might have an important relationship to the toxæmia of pregnancy. But Rosenstein, in 1909, established the fact that *fresh* human placenta did not contain any blood-pressure raising principle, and that the pressor substance described by Dixon and Taylor was a product of initial putrefaction. Rosenstein stated that the autolytic enzymes of the placenta could not produce pressor substances without the help of micro-organisms Several pressor bases, however, were developed in putrefying placenta from tyrosine. When placental tissue was extracted with equal parts of absolute alcohol, no pressor substances were developed; they were insoluble in alcohol. If, however, portions of a placenta could undergo autolysis during intra-uterine life, then these powerful blood-pressure raising bodies might be formed, and here there might be a sidelight thrown on the nature of eclampsia. The same pressor substance as was developed in dead placenta was also found in putrid meat. Moreover, Barger and Dale had shown that the pressor principle of ergot was due to the same body, which, curiously enough, was almost identical with adrenalin in its chemical formula. Tyramine—the chief active constituent of *watery* extracts of ergot, and used as a hypodermic preparation of that drug—was produced by

bacterial action on tyrosine. The pressor base of putrid meat or placenta was soluble in water but not in alcohol, and one had to bear that in mind when working experimentally with placental extracts.

Dr James Ritchie said he was not only struck with the great clearness of Dr Johnstone's descriptions, but with his fairness and unbiassed mind; because he got a negative result where he might have brought out, in some degree, a positive one. One so often read papers where the result appeared to be negative to the reader while the recorder declared it was positive.

Dr Johnstone, in replying, thanked the Society for the patience with which they had listened to his paper and the remarks they had made. One had a certain amount of reluctance in bringing forward a negative paper, but certainly after what had been said that night he felt encouraged to go on.

V. ACUTE DILATATION OF THE STOMACH AS A POST-OPERATIVE COMPLICATION IN ABDOMINAL CASES.

By E. Scott Carmichael, M.B., F.R.C.S., Assistant Surgeon, Hospital for Women, Edinburgh.

About three years ago I operated upon a married woman, æt. 30, for fixed uterine retroversion, and suspended the uterus by Gilliam's method. The patient recovered satisfactorily from the operation, but on the third day began to vomit, and continued to do so until the seventh day.

During these four days the patient was extremely ill, and I believed her to be suffering from delayed chloroform poisoning. There was some distension of the abdomen, but no tenderness or rise of temperature to suggest a generalised peritonitis. A very good observant nurse while attending to the patient rolled her over on her left side, and somewhat on to her face, with

the result that the patient vomited a large quantity of bile-stained fluid, in all from 2 to 3 pints as far as could be measured.

The patient was immediately relieved and never was sick again, making an uninterrupted recovery.

This case made a profound impression upon me, and I was unable at the time to explain the cause of this sequence of events, but considered the condition to be one of chloroform poisoning.

On 10th June 1910 I was asked by the house surgeon of Ward XI., Royal Infirmary, to see a patient operated upon by Mr Wallace nine days previously for hydatid cyst of the liver.

The history between the date of operation and the time when I was asked to see her was as follows:—

On 2nd June, the day after operation, her sickness became frequent, and continued so on the following days. Some distension of the abdomen was noticed on 4th June. The vomiting continued uninterruptedly until the morning of 10th June, when the house surgeon, suspecting peritonitis, called me in in Mr Wallace's absence.

The patient was markedly emaciated and looked like a starved woman. She was markedly dyspnœic and in great distress. On examining the abdomen, which was distended, one noticed that the distension was not uniform, but was limited especially to the epigastrium and left half of the abdomen as far as the hypogastrium. There was very slight tenderness, and the abdomen was soft, unlike a generalised peritonitis. On palpating more particularly distinct splashing sound could be detected, and there was marked dulness over the area mentioned. Believing the swelling to be due to a dilated stomach full of fluid, a stomach tube was passed and 5 pints of bilious, straw-coloured fluid drawn off. The patient experienced the greatest relief, and almost immediately fell off to sleep.

About 6 o'clock the same evening I was again rung up by the house surgeon, who said that the vomiting had recommenced in the course of the afternoon, and the patient was again very ill.

The stomach had again filled up almost to the extent which it had done in the morning. The patient was very weak, and rather than expose her to the risk of her atonic stomach filling again, I decided to open it. I therefore performed gastrostomy under ether, removing at the same time 4½ pints of fluid similar to that vomited in the morning.

At operation the stomach was found to be a flaccid bag, completely atonic, extending into and filling the pelvis and left iliac fossa, the small intestines being crowded into the right side of the abdomen. The dilatation extended into the duodenum, as far as the duodeno-jejunal flexure, the rest of the small intestine being completely collapsed. There were no signs of adhesions or obvious mechanical cause for the obstruction, the duodenum, however, being about three times its normal size and forming with the stomach one continuous sac. After emptying the stomach it contracted visibly down to almost its normal dimensions. The patient was much relieved, but died of exhaustion somewhat suddenly five hours after operation.

A post-mortem examination on the following day by Dr Shennan revealed the stomach and duodenum almost normal in size and no obvious cause to explain the condition.

On 29th December Dr Haultain removed a cyst-adenoma of the ovary in a woman, æt. 42, at the Women's Hospital, the tumour being the size of a six months' pregnancy.

The patient recovered from the operation satisfactorily, having no sickness on the day of the operation and none on the following day till 5 P.M., when she vomited (continuously) at short intervals. This continued for the next two days incessantly, the patient retaining nothing in her stomach.

On physical examination there was some slight distension in the upper part of the abdomen. Succussion of the stomach elicited splashing, the greater curvature of the stomach apparently descending below the level of the umbilicus.

On 2nd January a stomach tube was passed and $1\frac{3}{4}$ pints of bilious, straw-coloured fluid removed. The sickness ceased immediately, although she had vomited continuously for three days previously. Two days later, 4th January, in the afternoon, she vomited a pint of greenish, bile-stained fluid, which came up apparently without effort. She had no attempt at sickness until her discharge on 25th January.

These three cases are interesting as they illustrate various degrees of severity of a condition which I believe is frequently overlooked, especially in its milder forms. Many days of sickness or discomfort to the patient result from its non-recognition, and in severe cases it is not infrequently fatal.

On analysing more particularly the symptoms of this condition, the chief factors predominant are gastric atony or paralysis and hypersecretion.

In those cases reported by Bloodgood, Finney, Heile, and others, the patient not infrequently presents no symptoms for two days after operation. During this period in the more subacute cases the stomach is probably slowly dilating. Although the condition in most cases reported is one in which the dilatation affects the duodenum as well as the stomach, the latter organ, according to most authorities, is the primary site of the dilatation. No evidence of obstruction at the pylorus has been demonstrated, nor has there been any evidence of obstruction at the third or fourth part of the duodenum. The dilatation extends for the most part up to the duodeno-jejunal flexure, although cases are reported in which it may stop in any part of the duodenum. These facts seem to point against the view that the lesion is a gastro-duodenal mesenteric ileus due to kink at the duodeno-jejunal flexure, to dragging of the

enteric mesentery, or to pressure by the superior mesenteric vessels. This view is held by Brann and Leidel, who have made an exhaustive experimental study on the subject, but the conditions found at operation seem to disprove their theories.

The chief etiological factor seems to be some condition which produces paralysis with hypersecretion. The condition is not necessarily post-operative, as it has been observed to complicate eclampsia and puerperal septicæmia.

It is of the highest importance to recognise the condition early. In all cases of chloroform sickness persisting beyond the second day, the possibility of this condition should be eliminated. If sickness commences on the second or third day after operation it is highly suggestive of this condition.

The character of the sickness is, as far as I have seen in the two cases under my own observation, distinctive. There is not the severe retching which is so often characteristic of delayed chloroform poisoning, but the fluid is emitted more like the overflow of urine from a distended bladder, and seems more to well up into the mouth. The fluid is intensely irritating, and frequently irritates the skin of the lips and cheeks to the point of eczema.

Treatment.—If recognised early practically all these cases may be cured by passing the stomach tube, which however may require to be passed repeatedly. In late cases where the condition has led to starvation of the patient, gastrostomy, jejunostomy, and gastro-enterostomy have been performed. Certain it is that where the stomach lies like a stagnant, flaccid bag, filling up the greater part of the abdomen, drainage by one or other of the above means seems to be the *dernier ressort*. As to which of these is the best I have no experience, and it is not the purpose of this paper to discuss. In my own case I performed gastrostomy, which leads to contraction of the stomach and duodenum, and affords a means of feeding as well

as of draining the stomach, at a time when nourishment is of the greatest importance.

I am indebted to Dr Haultain and Mr Wallace for permission to refer to the cases operated upon by them.

The President said they would be glad to hear any remarks on this interesting and practical paper.

Dr A. H. F. Barbour said he had been very much interested, but he was not quite sure about gastrostomy being the treatment.

Mr Carmichael said he did not think cases should come to that, they should be treated before.

The President said he thought the paper one of considerable importance. There could be no doubt, unfortunately, that they seemed to get sickness of a much more mysterious type after abdominal operations than after any other, and sickness formed a very serious complication in many instances. Anything that could be done to help them to eliminate the sickness or minimise it would be thankfully received by all abdominal operators. He had himself seen several cases of acute dilatation, in fact almost a chronic dilatation, such as Dr Carmichael had described. In one of his cases an absolutely acute dilatation occurred within an hour or so and caused great collapse; in another case the patient died within two hours. What was the exact reason of this extraordinary sickness in abdominal section as compared with other operations, he could not say. He thought Mr Carmichael should be thanked for bringing before the Society one of the possible reasons of this condition, and also for showing how, in certain instances, it might be treated. Operators were frequently at their wits' end to stop the sickness.

Dr R. Johnstone asked if Mr Carmichael had tried washing out the stomach with adrenalin. It seemed to him that if

there was a hypersecretion, as in the chronic cases, adrenalin might prevent it.

Dr Oliphant Nicholson asked if he had tried pituitary extract.

Mr Carmichael said he had not tried adrenalin, but he thought it a very valuable suggestion; it might have a tonic action on the muscular coat of the stomach, as well as an inhibitory action on the secretion. He had tried pituitary extract and found it distinctly useful.

VI. *Dr A. H. F. Barbour* and *Dr B. P. Watson* read the following papers:—(*a*) ON THE CYSTIC OVARY; (*b*) TUBERCULOUS PYOSALPINX.

(*a*) THE CYSTIC OVARY.

By A. H. F. Barbour, M.D., LL.D., F.R.C.P.E., Lecturer on Gynæcology, University of Edinburgh, Gynæcologist to Royal Infirmary, Edinburgh; and B. P. Watson, M.D., F.R.C.S.E., Lecturer in Midwifery and Gynæcology, School of Medicine of the Royal Colleges, Edinburgh.

This communication takes the form of a demonstration rather than a discussion of pathological changes. It falls into two parts, of which the first, dealing with changes in the ovary, may be dealt with in a few words. The so-called cystic ovary used to be described as the result of inflammatory changes within the Graafian follicle—follicular ovaritis. The correct view, however, is that these cystic ovaries are the result of non-rupture of Graafian follicles. This may be due either to a change in the follicle itself, after it has attained a certain degree of dilatation which prevents it from maturing and rupturing, or to a sclerotic condition of the tunica albuginea, resulting from a previous inflammatory condition of the surface of the ovary. This thickening of the albuginea may be

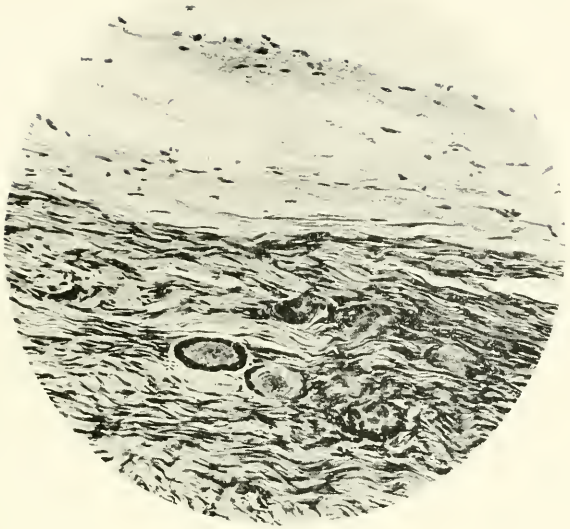


FIG. 1.

FIG. 2.

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sufficient to prevent the rupture of the normal follicle. The result is that the cells of the zona granulosa continue to secrete, so that a small retention cyst is formed. A simple serous cyst is thus produced. The wall of such a cyst consists of fibrous tissue arranged circularly and a lining of several layers of the zona granulosa cells (see Plate, Fig. 1). In many such specimens there is evidence of a tubal inflammation with matting of the tube and ovary and inflammation on the surface of the latter (see Plate, Fig. 2). The tubal infection is probably the primary one; the surface of the ovary is secondarily affected and leads to the formation of the cysts. In a more acute process there may be organismal infection of the stroma of the ovary and of the cyst resulting in pus formation. In a number of follicles the content is blood or blood-stained fluid, and in the wall of many of these lutein cells are present. These are derived from atresic follicles which have failed to rupture, but in which the lutein formation has proceeded.

As to the length of time that this condition takes to develop, we are slowly accumulating data which can only be got from patients operated on a second time. In this connection this specimen is of interest, removed from a patient who developed symptoms calling for the removal of the second ovary, which was ascertained to be healthy on a previous operation eighteen months before.

Such ovaries are not stages in the production of ovarian tumours, and ought not to be classified along with them. Their removal is a question of expediency, and is determined by the amount of disturbance they give rise to. It is doubtful whether the pathological condition calls for removal of diseased tissue unless there are distinct symptoms. It will be an interesting subject for discussion whether the resection of the cystic portions of a diseased ovary is called for in these cases.

(*b*) TUBERCULOUS PYOSALPINX.

By A. H. F. Barbour, M.D., LL.D., F.R.C.P.E., Lecturer on Gynæcology, University of Edinburgh, Gynæcologist to Royal Infirmary, Edinburgh; and B. P. Watson, M.D., F.R.C.S.E., Lecturer in Midwifery and Gynæcology, School of Medicine of the Royal Colleges, Edinburgh.

The whole question of tuberculosis of the tube is an interesting and important one. It is frequently found on opening the abdomen that tuberculous tubes are present with no evidence of the presence of tuberculosis elsewhere in the pelvic or abdominal cavities. Or it may be found that in general abdominal tuberculosis the tubes are extensively affected and have apparently been the primary focus of disease. Whether the condition is really primary in the tube or not is a very difficult question to decide, and as yet no definite conclusion has been arrived at. It is difficult to exclude a blood or lymphatic infection from a distant focus, say, in the lung or in mesenteric glands. Whilst we cannot give any opinion on this point, the routine examination of three cases of large tuberculous pyosalpinx has given interesting results, which we propose to illustrate.

Case I.—Mrs B., æt. 36, nullipara. For nine years the patient has had a dull pain in the abdomen specially marked in the right iliac fossa. During the last four years these pains have been more severe, with shorter intervals between the attacks. For the past three years she has noticed a swelling in the abdomen situated apparently as she describes in the middle line. Her general health has been good, and she has not got thinner of late years. Menstruation has always been irregular; intervals varying from five to eight weeks, duration three days, amount of loss moderate. There were seven weeks' amenorrhœa previous to admission. For the past two years there has been a yellowish leucorrhœa with no odour. There have been no pregnancies. She is a well-nourished, healthy

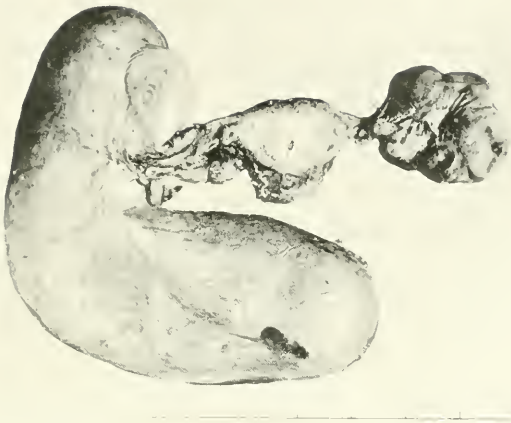

FIG. 1.

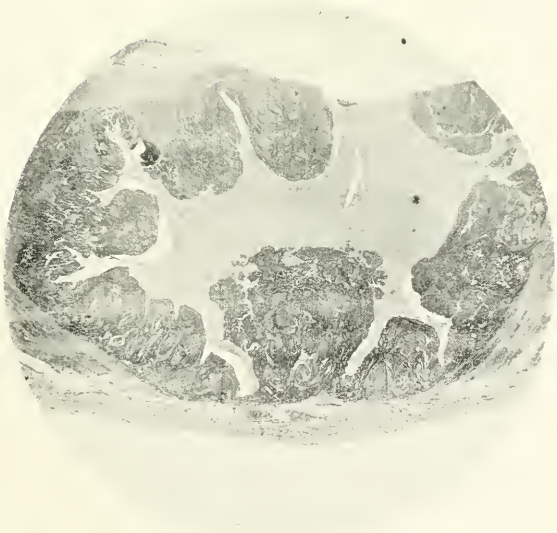

FIG. 2.

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woman with abundant subcutaneous fat. There is no temperature.

The abdomen is distended to the size of a six months' pregnancy. On palpation a rounded swelling is felt, filling up the lower part of the abdomen and reaching almost to the umbilicus. This swelling is dull on percussion and semi-fluid to feel. On vaginal examination the os is nulliparous, pointing backwards, and bi-manually the uterus can be felt through the anterior fornix freely movable and not enlarged. A tense cystic swelling can be felt filling up the fornices of the vagina and extending above the brim of the pelvis behind the uterus.

On opening the abdomen a tumour the size of a small melon was felt extending out of the pelvis. It was folded on itself and on tracing it downwards was found to be a dilated tube. The isthmus was partially twisted but was undilated. The other tube was also distended, but to a lesser degree, and lay below the right tube towards the pouch of Douglas. There was an omental adhesion to the larger tube, and adhesions were present between each tube and the corresponding ovary. Both tubes and ovaries together with the uterus were removed, the latter being amputated at the level of the isthmus. The peritoneum of the pelvic floor was stitched in in the usual way, and the abdomen closed. The patient made an uninterrupted recovery.

The specimen consists of the uterus amputated at the level of the isthmus together with both tubes and ovaries (see Plate I., Fig. 1).

The left tube is tortuous, dilated in the ampullary and infundibular regions, and the fimbriated end is occluded and adherent to the ovary. The isthmic portion is of normal thickness. The whole tube measures 6 inches in length and 3 inches in circumference at its widest part.

The right tube is enormously dilated in the ampullary and infundibular regions, forming a large tumour measuring 13 inches in length and 10 inches in circumference. The swelling is folded on itself, and there are peritonitic adhesions between the folds and also tags of adherent omentum. The

colour is greyish-red, and on the surface are slightly raised areas about the size of sixpence which are distinctly calcareous. There are no recent tubercle nodules on the surface. In consistence the swelling is semi-fluid, and on opening it the content is found to be glairy mucus rather turbid in colour.

Both ovaries are enlarged and cystic. The right is free from adhesions but the left is adherent to the tube, and is covered with adhesions which had been separated from the abdominal structures. The uterus is slightly enlarged, contains no visible nodules or tubercles, and has no adhesions.

Microscopic Examination.

Left Tube.—Part next uterus only slightly dilated. On transverse section it is seen that the mucous membrane is greatly thickened by a hypertrophy of the papillary folds. These latter, instead of consisting of a simple core of connective tissue covered with a single layer of columnar epithelium, are large expanded masses of connective tissue containing numerous giant-cell systems and tubercle nodules (see Plate I., Fig. 2). The epithelial covering has been lost in places, but in the midst of the connective tissue large areas of it are present. It is in places arranged in the form of gland acini, and in others in solid strands and clumps. The simple acinous arrangement is more marked in the deeper parts next the muscular wall, the more solid and irregular arrangement in the centres of the projections and towards the tips. In this latter region also the tubercle nodules are more numerous and the necrotic process more marked. The muscular wall is in most places normal except for some degree of thickening. Only in a very few areas is it invaded by tubercle nodules, and these areas closely abut on regions where such nodules are present in the deep part of the mucosa. The content of the tube is a clear homogeneously staining material with a few cells in it next the mucous lining. On higher magnification it is seen that the

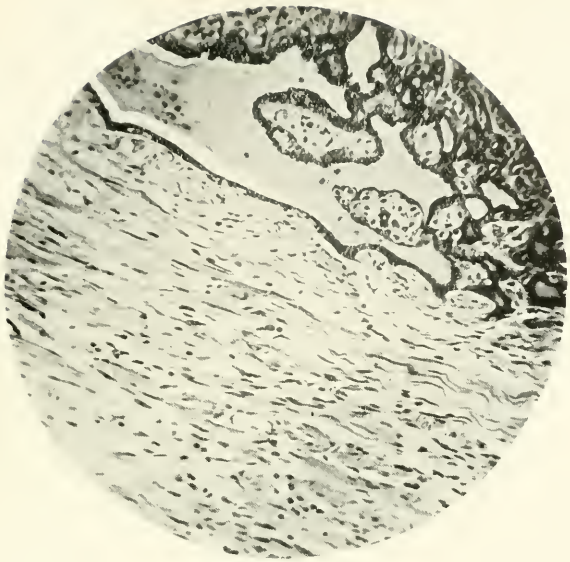

FIG. 1.

FIG. 2.

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acinous-like arrangement in the deep part of the mucosa next the muscular wall is due to the branching and hypertrophy of the villous processes and their section at different angles. The connective-tissue cores of these processes can be distinguished and the covering of a single layer of epithelium. At places this epithelium dips into the muscular wall more deeply than usual in the form of tubular prolongations (see Plate II., Fig. 1.) The connective tissue is more or less normal in appearance, except for a slight cellular increase, and in only a few areas is it invaded by the tubercle nodules. The epithelial covering is in a single layer and normal in appearance. On tracing these papillary folds outwards they expand into large masses of tissue, neighbouring processes apparently fusing with each other. Tubercle nodules with giant-cell systems are very numerous, and between these are the epithelial masses already referred to (see Plate II., Fig. 2). So closely are these packed that there is practically no healthy tissue left between them. The tubercle nodules are of the ordinary type, consisting of the so-called epithelioid cell and giant cell, and outside these collections of round cells with large nuclei. They vary greatly in size. Some of them are necrotic in the centre, and at the tips of some of the processes there is a distinct invasion with polymorpho-nuclear leucocytes indicating a mixed infection. In only one or two places can the nodule be seen breaking through the surface of the mucosa and discharging its content into the lumen.

The appearances of the epithelium are interesting. In the deeper parts next the muscular wall, where it forms a uni-cellular layer covering the bases of the villous processes, it is flatter than normal and extends more deeply into the muscular coat, and cilia seem to be absent. Here and there a connective-tissue cell can be seen making its way between the individual cells, but these are not frequent. At the tips of some of the processes in which there is no tubercular invasion, the epi-

thelium is proliferating rapidly, forming several layers, and being cast off into the lumen. In these processes the vessels are dilated, and there is active proliferation among the connective-tissue cells of the part. In one such villous process the epithelium is everywhere in this condition, except at one point where it is degenerated and broken down, and at that area is a tubercle nodule extending into the connective-tissue core. From the surfaces of the fused processes containing many tubercle nodules, the epithelium has to a large extent disappeared, and it is in these that the masses of included epithelium are seen (see Plate II., Fig. 2). In the deeper parts, next the muscularis, it is arranged in an acinous-like way, the appearance being probably due to the cutting in different directions of much branched epithelium-covered processes, and to the fusing of the tips of these processes, the bases remaining separate. Nearer the surface this acinous arrangement is lost, especially in those areas where the tubercle nodules are numerous. In these regions the epithelium forms strands and masses many cells thick, giving on superficial examination an appearance very like a carcinoma (see Plate II., Fig. 2). There is, however, no evidence of an active proliferation and invasion; rather the reverse, for everywhere it can be seen that these epithelial masses are being invaded by round cells with large nuclei which are making their way in between the individual cells and destroying them. Round each of these cells a clear space is usually visible, probably due to shrinking of protoplasm, and beyond that the remains of several epithelial cells arranged circularly like a minute gland acinus. These are, in turn, invaded by other cells from the periphery, and so the masses of epithelium are broken up, degenerate, and disappear. This process is most active towards the surface of the mucosa where the tuberculous process is most advanced.

Left Tube—more dilated Outer Portion.—The muscular wall is thinner and there are no tubercle nodules visible in it. The

FIG. 1.

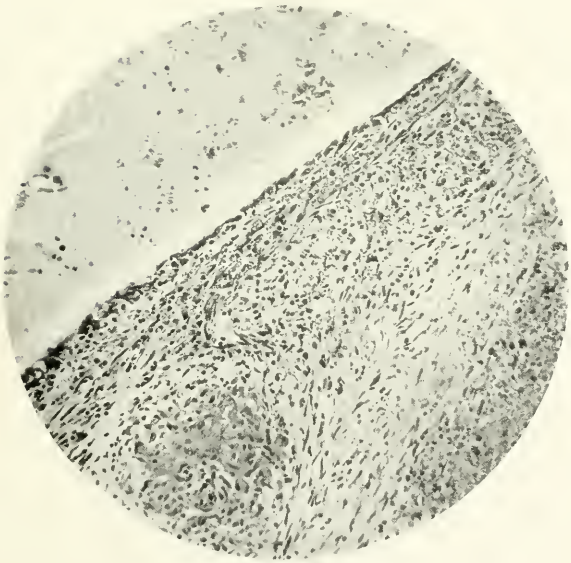

FIG. 2.

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mucous membrane is thinner than in the less dilated portion and the surface is more regular, the villous projections having almost entirely disappeared, owing to the breaking-down of their free extremities. Next the muscular wall there is a continuous covering of a single layer of columnar epithelium, and springing from this surface the bases of the villi can be distinguished still unfused, although the bodies of the processes nearer the lumen have fused together to form a solid mass of tissue (see Plate III., Fig. 1). In this latter is included epithelium, for the most part in a state of degeneration and much invaded by round cells. Between the epithelial masses are tubercle nodules and masses of tuberculous granulation tissue, which is everywhere breaking down on the surface and being cast off into the lumen. The tuberculous tissue is evidently breaking down and disappearing, leaving next the muscularis a healthy layer of epithelium and submucous coat as yet uninvaded by the tuberculous process (see Plate III., Fig. 1).

Right Tube—greatly dilated Outer Part.—In sections of this tube a still further stage of the process can be seen. The lining of the lumen is smooth, and is covered with a single layer of epithelial cells. The submucous tissue is finely cellular, contains some extravasated blood, one or two tubercle nodules, and some islands of included epithelium (see Plate III., Fig. 2). It has all the appearances, except for the one or two isolated tubercle nodules, of healthy granulation tissue.

Sections of the uterine wall fail to reveal any evidence of tubercle.

Case II.—M. N., æt. 30, school teacher, nullipara, was admitted to Ward 35, Royal Infirmary, on 14th March 1910. Eight or nine weeks previously she had noticed a swelling of abdomen, but had nothing else to complain of. No family history of tubercle except that one brother died of meningitis. Menstruation of four-weeks' type, duration four days, slight

dysmenorrhœa. No amenorrhœa. Has always had slight leucorrhœa. Occasional pain on micturition.

Patient is thin. A swelling is seen in abdomen reaching midway between pubes and umbilicus. It is semi-solid, is movable from side to side and vertically, and is dull on percussion. Cervix is small and nulliparous and directed forwards. In the posterior fornix is a mass continuous with that felt through the abdomen. Through the right fornix a sulcus can be felt in the swelling, the part above being globular and seeming to move apart from the swelling below.

On opening the abdomen the left tube was found greatly enlarged. It extended from the left angle of the uterus towards the left, then turned on itself with a complete twist, and becoming greatly dilated ran to the right across the pelvic inlet. It again turned on itself, became more dilated, and ran towards the left side. The folds were adherent to each other and to the omentum and intestine. These were separated and the tube removed. It was then found that the right tube was in a similar condition, lying low down in the pouch of Douglas and adherent. It was removed and, as there was a large raw area left, the uterus was also removed. She made a good recovery, and was in good health some months later.

The specimen consists of both tubes and ovaries and the uterus (see Plate IV., Fig. 1).

Both tubes are equally enlarged by dilatation of their ampullary and infundibular regions, measuring 13 inches in length and 10 inches in circumference. They are bent upon themselves, and numerous dense adhesions bind the folds together. The fimbriated extremities are occluded and are densely adherent to the ovaries, which are enlarged and cystic. Numerous adhesions are present over the surface of the swellings, especially on the anterior aspect of the right tube. They are semi-fluctuating in consistence. On section of the uterine end the tube wall is thickened, but the lumen is not dilated. In the dilated portion in the ampullary and infundi-

FIG. 1.

FIG. 2.

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bular regions the content is muco-pus and the tube wall is thinned.

Microscopic Examination.

Less dilated Part of Tube.—The muscular wall is thinner than usual but otherwise normal. The mucous membrane is enormously thickened by the swelling and fusion of the villous processes, and by the invasion of these with tubercle nodules (see Plate IV., Fig. 2). This invasion extends deeper down towards the muscular wall than in the preceding specimen, and the result is that the origins of the individual villous projections cannot be distinguished in many areas. In places these can, however, be made out, and in these areas many villi are quite unaffected even at the tip. The included epithelium has more of the acinar arrangement than in the previous case, there not being so many clumps of epithelial cells present. This epithelium is being invaded and broken up by round cells, especially towards the free surface. Here, also, the tuberculous process is most advanced, and numerous caseating areas are present.

In general features, therefore, the picture here is the same as that present in the less dilated portions of the tube in Case I.

More dilated Part of Tube.—The muscular wall is thinned, and the mucous membrane is greatly reduced in thickness as compared with that of the less dilated part. The mucosa has lost its villous projections except for an occasional one here and there. The included epithelium has in most places entirely disappeared, except in the few villous processes still left where the acinar arrangement is still seen. The tissue consists of breaking-down tubercle nodules, and a great infiltration with small round cells and polymorphonuclear leucocytes. It is everywhere ulcerating and breaking down on the surface, the débris accumulating in the lumen (see Plate V.).

There is here again, then, evidence of a mixed infection

with ulceration and breaking down of the thickened mucosa, and it presents an intermediate stage between the two areas described in Case I. (see Plate III., Figs. 1 and 2).

Uterine End of Tube—not dilated.—The lumen is normal in size, and as is usual in this part of the tube the villous processes of the mucous membrane are few and small (see Plate VI., Fig. 1). There is no invasion of the mucous membrane with tubercle, but there is some proliferation of the epithelial covering and of the connective tissue, giving at places an acinus-like arrangement on section. The muscular wall is thick. The submucous and inner muscular layers contain numerous tubercle nodules studded all round the lumen, and in these areas deep in the tube wall are spaces, like gland acini, lined with columnar epithelium (see Plate VI., Fig. 2).

Uterine Wall.—In the mucous membrane near the tube angle one or two distinct tubercle nodules are present (Fig. 10B).

Case III.—M. M., aged 27, single, nullipara.—The patient was treated in Ward 35 seven years ago for a condition which, on admission, resembled a subacute appendicitis, but which later developed into what was described as a pelvic cellulitis on the right side. At this time there was a hardish swelling felt through the right fornix. She had irregular rises of temperature. The condition completely cleared up under treatment and she left the Ward apparently cured. Since that time she has been in good health until three months before admission, when she began to have a profuse yellow leucorrhœal discharge, accompanied by inflammation of the external pudenda and pain on micturition. This was followed by severe abdominal pain which rendered her unfit for work, and she had been in bed for a fortnight before admission. One brother and one sister had died of consumption. Menstruation is regular, of twenty-eight-day type, rather profuse in amount. Dysmenorrhœa of a pre-menstrual type has always been present.

On abdominal examination nothing is found. *Per vaginam*

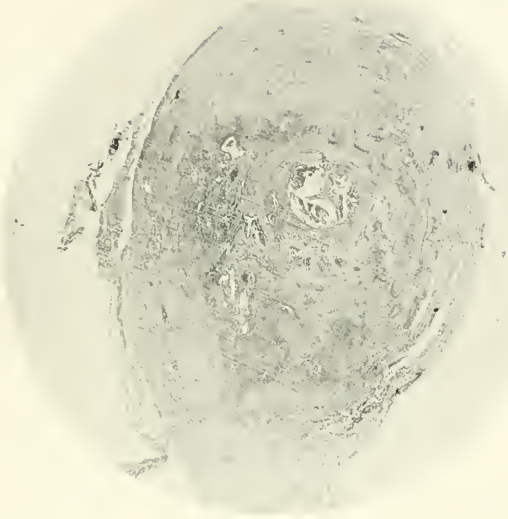

FIG. 1.

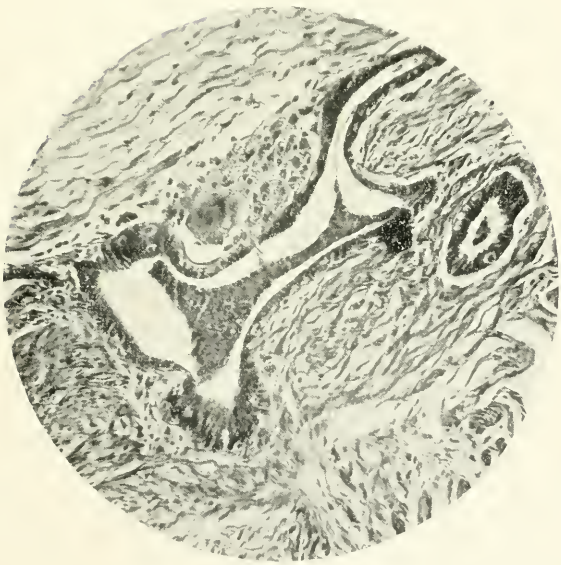

FIG. 2.

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the cervix is nulliparous and looks downwards and backwards. The uterus is lying to the front, not enlarged and not tender. To the right of the uterus a swelling is felt coming close up to it. This swelling is fixed, somewhat tender, and semi-solid in consistence. It fills up the right and part of the posterior fornices. An exactly similar swelling is present on the left side.

On opening the abdomen it was found that the swellings were distended tubes, each about the size of a goose's egg. There were adhesions between each tube and ovary, but none to the intestine. The infundibulo-pelvic ligament on each side was clamped, and the undilated isthmic part of the tube similarly dealt with. Both tubes and ovaries were then removed. There was no evidence of any peritoneal infection, and the abdomen was closed without drainage. The patient made a good recovery and left the Ward three weeks later. When seen a month afterwards, a small lump about the size of a cherry was felt in the situation of the right stump. The patient still complained of the profuse leucorrhœa. Two months later she had put on a stone in weight and was feeling very well.

The specimens consist of both tubes and ovaries, the former dilated into retort-shaped swellings, measuring $7\frac{1}{2}$ inches in length and $7\frac{1}{2}$ inches in circumference.

The tubes are practically identical in appearance (see Plate VII., Fig. 1). The isthmic portion is only slightly thickened, but just beyond it is a rounded swelling the size of a cherry growing from the upper aspect. On section this swelling is found to be an abscess in the tube wall and excentric to the lumen. Beyond this the tube becomes gradually dilated, reaching its maximum size in the infundibular region. The fimbriated end is closed and adherent to the ovary, which is large and cystic. There are numerous adhesions on the under aspect of the tube, but none on its upper aspect except at the isthmic portion. The consistence is fluid and the content is thick greenish-yellow pus.

Microscopic Examination.

Inner End of Tube slightly dilated.—The lumen is slightly dilated and the mucous membrane is hypertrophied. The folds of mucous membrane are thick and contain many tubercle nodules and included epithelium, mostly in an acinus arrangement, but also in solid masses. The epithelium is absent on the surface of the projections. In the substance of them it is in most places undergoing degeneration and is being invaded by phagocytic cells. The tuberculous nodules extend into the inner muscular layer, causing destruction of the muscle. In the vicinity of these areas of invasion the epithelium also dips deep into the muscle in the form of tubular prolongations. The tuberculous invasion is as marked in these regions as at the tips of the villous processes, the basal mucosa being in places entirely destroyed.

Dilated Part of Tube.—The muscular wall is thin. The mucous membrane has entirely disappeared, and in its place is a thin layer of granulation tissue with here and there included epithelium and giant cells.

Small dilated Part near Uterine End.—The dilatation is due to the presence of a tuberculous abscess in the tube wall. The lumen of the tube is intact and the mucosa is unaffected by tubercle. In the outer muscle coat an abscess has developed. It is lined with tuberculous granulation tissue, and in several areas there are calcareous deposits (see Plate VII., Fig. 2).

Conclusions.

These three cases resemble each other closely in their clinical and pathological manifestations. In all there was an absence of symptoms pointing to any active or extensive tuberculous process proceeding in the abdomen. In two there was a history of tubercle in the family, but in neither case was this marked. In two attention was directed to the condition

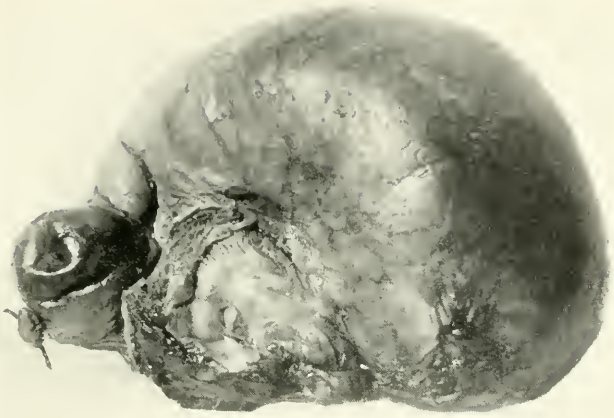

FIG. 1.

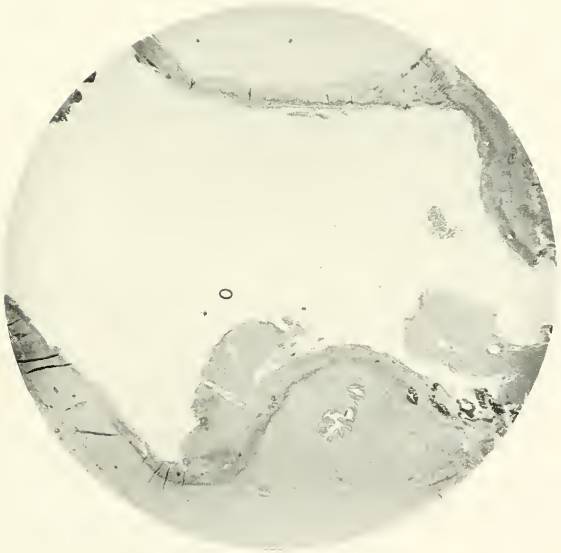

FIG. 2.

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by the appearance of an abdominal swelling, and in the third a leucorrhœal discharge, possibly gonococcal in origin, led to an examination of the pelvis and the detection of the swellings beside the uterus. In all three the condition was bilateral. The condition of the tubes in the first two is very similar—great dilatation of the ampulla and infundibulum and only slight thickening of the isthmus. In the third case the dilatation had not proceeded to the same extent, and in it the content of the tubes was more distinctly purulent than in the other two. In none of the cases was there any evidence of tubercle in the abdomen, or elsewhere.

There is the same constancy in the microscopic appearances. In the less dilated parts of the tubes there is a great thickening of the mucous membrane produced by a hypertrophy and fusion of the folds, as the result of the development in them of tubercle nodules (see Plate I., Fig. 2, and Plate IV., Fig. 2). The tubercle nodules are entirely confined to the mucous membrane, and are most abundant in the superficial parts of it. The bases of the folds and the submucous tissue and muscularis are healthy. As the result of the irritation of the tuberculous poison there has been a hypertrophy of the epithelium resulting in a deeper penetration of it into the muscular layer, and to the formation of strands and masses of it in the substance of the mucosa. This hypertrophy has been so great as to give to this part of the tube an appearance very like carcinoma (see Plate II., Fig. 2). Following on the overgrowth there has been a degeneration and destruction of the epithelium as the tuberculous process advanced. The masses have become broken up by the invasion of phagocytic cells, and all stages in the process of disintegration can be traced. The initial hypertrophy has resulted in an increased secretion, and apparently in an alteration in the character of the secretion, so that the lumen of the tube and the spaces formed from the fusion of neighbouring processes have become filled with a mucoid material.

In the more dilated parts of the tube at the outer extremities the tuberculous process is more advanced. Ulceration has occurred, causing a destruction of the superficial parts of the mucous membrane, so that the villous projections are no longer visible (see Plate III., Fig. 1, and Plate V., Fig. 1). The epithelium has almost completely degenerated, except in the immediate vicinity of the muscular wall, and all that is left is a lining of tuberculous granulation tissue, while the cavity is filled with débris and muco-pus. There is evidence in parts of all the specimens that there is an acute inflammatory process also present pointing to a mixed infection.

In the most dilated part, namely, in the wall of the large pyosalpinx of Case I., a still further stage is seen—the stage of natural cure. The mucous membrane has been entirely cast off, leaving only a covering of a single layer of flattened epithelium, underneath which is a healthy, newly forming tissue with only here and there an isolated tubercle nodule (see Plate III., Fig. 2). The muscular wall is healthy.

At the extreme uterine ends of the tubes a different picture is presented. The mucous membrane is free from tuberculous infection, and shows only a hyperplasia of the epithelial covering and a deeper extension into the muscular wall, resulting in the presence of gland-like spaces lined by epithelium in the muscular wall at a considerable distance from the lumen (see Plate VI., Figs. 1 and 2). In the muscular wall and submucous tissue are numerous tubercle nodules. In one of the specimens (Case III.) the tuberculous process in this situation has extended to such an extent as to lead to the formation of an abscess in the wall (see Plate VII., Fig. 2).

It would thus appear that the tuberculous infection is most advanced in the outer dilated parts of the tubes, and is in that situation confined to the mucous membrane, which is entirely destroyed by it. The muscle wall is not affected. In the less dilated part of the tube near the uterus, the destruction of

tissue is less marked and the infection is still confined to the mucous membrane, and is most advanced in the superficial parts of it. At the uterine end of the tube the mucous membrane is unaffected, but tubercle nodules are present in the tube wall. In Case II., tubercle nodules are also present in the mucosa of the uterus in the neighbourhood of the tube angle.

These are the facts. What deductions can we draw from them? Has the infection of the superficial parts of the mucous membrane in the outer region of the tube been a direct infection through the surface, or has it come by the blood-stream? To this we can give no definite answer, but it seems to us that the appearances strongly suggest that it is a case of direct infection through the surface. When the physiology of the tube is considered this appears not unlikely. There must be, by the ciliary action in the healthy tube, a constant suction at its outer end which will draw into it any small bodies such as tubercle bacilli from the peritoneal cavity. In none of these cases was there any evidence of abdominal tuberculosis, but it is conceivable that the organisms were there in small numbers, derived from some focus in the lungs or in mesenteric glands. Those that remained in the peritoneum may have been destroyed, for it is well known that the peritoneum can deal with small doses of tubercle bacilli. Only those which reached the anterior of the tube may have continued to grow. It is probable that this method of infection of the tube from the peritoneal cavity takes place in other conditions, such as appendicitis, and it seems to us not unlikely that the same thing may happen in tubercle.

After the primary infection of the mucosa of the outer part of the tube, there is evidently a lymphatic spread inwards towards the uterus; and that results in tuberculous deposits in the tube wall near the uterus, and in the uterus itself.

Another point of interest in these specimens is the reaction

of the epithelium to the irritation of the tuberculous poison. It is first of all stimulated to overgrowth with deep extension into the tissues, giving an appearance very like carcinoma. Later it undergoes destruction. It has been held by many writers that primary carcinoma of the tube is secondary to chronic inflammation, and looking at these sections this appears possible. It is conceivable that an arrest of the tuberculous process before destruction of tissue occurred might leave the epithelium in such a state of activity as to take on a cancerous character. This reaction of the epithelium has been described by several writers in cases of tuberculous pyosalpinx and in other chronic inflammatory affections of the tube.

This epithelial overgrowth is also seen at the uterine end of the tube where the gland-like spaces are present in the muscular wall. This is an appearance which has been described as adeno-myoma of the tube. It has been described by several writers in association with tubercle, and by most, including Von Franqué, it is regarded as secondary to it. With this we agree.

DESCRIPTION OF PLATES.

PLATE

I.—FIG. 1.—Double pyosalpinx removed along with the uterus (Case I.). The right tube is enormously distended.

FIG. 2.—Section of slightly dilated part of the left tube. Note the thickening of the mucous membrane and the presence of tubercle nodules in it.

II.—FIG. 1.—Left tube, slightly dilated part near inner end. The epithelium next the muscular coat is intact. Nearer the surface of the mucosa tubercle nodules are present and the epithelium is proliferating.

FIG. 2.—Left tube—section of mucosa from slightly dilated part near inner end. The epithelium is proliferating and forming solid masses between which are tubercle nodules. Note the invasion of the epithelial masses by round cells.

III.—FIG. 1.—Mucosa and tube wall from more dilated outer part. There is a complete epithelial lining next the muscular wall,

PLATE

which is healthy. Beyond this the mucosa is infiltrated with tubercle and breaking down.

III.—FIG. 2.—Right tube—outer dilated part. The surface of the mucous membrane is smooth and covered with a single layer of flattened epithelium. In the connective tissue are one or two isolated tubercle nodules. A process of cure.

IV.—FIG. 1.—Double pyosalpinx (Case II.). Both tubes are equally enlarged. Note their size compared with the uterus.

FIG. 2.—Inner slightly dilated part of tube. The mucosa is greatly thickened and contains numerous tubercle nodules. The epithelium is proliferated.

V.—Outer more dilated part of tube. The mucosa consists of a mass of breaking-down tuberculous granulation tissue.

VI.—FIG. 1.—Uterine end of tube—Isthmus. There is no tubercle in the mucosa. The tube wall is greatly thickened and contains many tubercle nodules. Note the presence of epithelium-lined spaces in the tube wall.

FIG. 2.—High-power view of one of the gland-like spaces in the muscular wall, as seen in Fig. 1. Note the tubercle nodule in relation to it.

VII.—FIG. 1.—Pyosalpinx (Case III.). The small rounded swelling in the region of the isthmus is a tuberculous abscess in the muscular wall.

FIG. 2.—Section through the rounded swelling at the isthmus, as seen in Fig. 1. The lumen of the tube is intact. The swelling is a tuberculous abscess in the tube wall. The dark areas are calcareous deposits.

The President said they would be glad to hear remarks on the excellent demonstration which they had just had. He thought one of the most important features regarding cystic ovaries which they had learned to-night—although some of them knew it before—was how to distinguish between a cystic tumour of the ovary and a simple dilatation. The clinical features were interesting. In a tumour they had a new growth which produced, apparently, no definite impression on the surrounding ovarian tissue, hence they were associated with little or no pain; that accounted for the painlessness of ovarian

tumours. The cystic ovary, on the other hand, in the majority of instances, was associated with very great pain. That, of course, might be accounted for by the fact that it was a more or less similar condition to an abscess. Also, occasionally, cystic ovaries were attended with pain which it was difficult to account for. From the clinical appearances one was almost led to believe that the most painful ovary was the cirrhotic ovary without cysts. Sometimes a combination was found. He (Dr Haultain) remembered one case where a second cyst occurred within eighteen months. Anything which tended to give an idea of the pathology of the ovary was necessarily of very great value, particularly from the naked-eye appearances; because from the point of view of the operator it was one of the most difficult things to know whether to remove an ovary or not. Conservative surgery of the ovary was practically in its infancy; so far, in his (Dr Haultain's) hands, it had been a complete failure. After puncturing cysts, or perhaps removing a little one, the ovaries had had to be removed; on several occasions he had had to remove the ovaries later, after this operation had been done by himself, or by some other operator. One did not like to remove the ovaries, but one rather depreciated an operation which left the patient as bad as before. On the other hand, if one found distinct cysts of considerable size, showing distinct lining membranes that could be recognised, then these cysts should be excised, and the ovary stitched up. He remembered two cases, one in particular in which he removed a large ovarian tumour and excised a cyst; after that the woman had two or three children, showing that the ovary was functionally active. There was no pain. Anything they could get in the way of pathological specimens or demonstrations such as they had had that night, which would give them information regarding the naked-eye pathology of the ovary, could not fail to be of the greatest value to operators as regards conservative surgery. The demonstration of the tuberculous

tube was extremely interesting. He had met with a number of tubes which had been universally thickened. He had never thought of the tubercle passing from without inwards. One would like to know where the tubercle attacked the tube first; or in what way the salpingitis was developed. Was the peritoneal inflammation usually secondary, or was the tubal condition secondary? The demonstration, he thought, must have been of very great benefit to the Society; personally he had learned much from it.

Mr E. W. S. Carmichael said he had listened with very great interest and had learned a great deal. The President had referred to the question of conservative surgery of the ovary. From his (Mr Carmichael's) very short experience of gynæcological surgery, he must say that they could do very little when cystic ovaries had to be dealt with. If there was great pain, and the patient came to the doctor on account of the pain, then he supposed there was nothing for it but to remove the ovary. One had frequently seen the ovary removed for cysts where no cysts existed. Cystic ovaries should not necessarily always be removed. If the condition was of follicular origin and the patient had not suffered prominently from pain, he did not remove the ovary. The length of time of development was interesting—eighteen months in Dr Barbour's case. He (Mr Carmichael) had had an interesting case in which a young sterile woman suffered from ovarian pain; the left ovary was extremely painful and he removed it. On examining the right ovary at the time, it was apparently healthy. Three months later the patient came back and said that the right was as painful as the left had been. Very much against his will he removed the right ovary and found it contained a cyst, about the size of a walnut, which had developed within three months. With regard to the Fallopian tube he was extremely interested, because he had always been inclined to think, from what he had read, that if there was a

double condition in the pelvis—peritonitis and salpingitis—the salpingitis was really the cause of the peritonitis in most cases. He had had one or two instances recently which had practically proved to his mind that one got a secondary infection in the tube from the peritoneum. He could not help thinking, from what Dr Barbour and Dr Watson had said, that tubercle of the Fallopian tube was an infection from the fimbriated end towards the uterus. In the Children's Hospital recently he had operated on an interesting case. The patient, a girl, æt. 10 or 11, was suffering from acute appendicitis and subacute peritonitis. The appendix was removed in the usual way, and the patient recovered. Three years afterwards he was asked by her doctor to see her for a tumour in the lower part of the abdomen. He found what he believed to be a cystic tumour; he operated, and found a pyosalpinx of the right Fallopian tube. That case was another example of infection from the peritoneum to the Fallopian tube. Pneumococcal peritonitis was less common in the female than in the male, and the usual reason given was that it was an extension from the Fallopian tube into the peritoneal cavity. He had had 11 cases of pneumococcal peritonitis; 7 had been in males and 4 in females. He was inclined to think that the infection spread upwards rather than downwards.

Dr James Young said he had been particularly interested in the second part of the demonstration, and more especially with regard to the point which had been brought out in connection with the proliferation of epithelium, which occurred in response to the stimulus of the tubercle bacillus. This point had been worked out previously and, he thought, in the tube. The active invasion resembled a cancer involvement. He had read somewhere that the proliferation of the epithelium might be so extensive and active as to be almost unrecognisable from active cancer. The same epithelial invasion had been ascribed to pneumococcal invasion of the tube. It was more apt to

occur in the pneumococcal or gonococcal than in the streptococcal and staphylococcal forms. He remembered showing before the Society appearances almost entirely analogous to those they had seen to-night. The epithelial involvement and invasion were so marked that, had one not known the condition, it would have been suggestive that the patient was actually suffering from cancer of the tube. The point was one of extreme interest; it seemed to him to touch the border line of questions which were concerned with the etiology of cancer. Here one had a stimulation of a material which possessed the power to excite an active proliferation which, in its microscopic appearances, was entirely analogous to cancer. It suggested that the gland-spaces which were found in the tubal wall, and described as an adeno-myoma, were due to exactly the same condition, namely, that the tubules had been cut across at a part somewhat distant from the central origin.

Dr Barbour, in replying, said the President had referred to the importance of getting information to guide the operator. They had suggested to them as a subject for discussion: Should the ovary be entirely removed, or was it justifiable to resect diseased portions of it? Two views had been advocated. The President seemed to be against resection; Mr Carmichael was in favour of being as conservative as possible. On the whole, he (Dr Barbour) was inclined rather to the President's view; he would rather either not touch the ovary, or he would remove the whole of it. With regard to pain, he was not sure that it was simply due to pressure; he thought it was due to involvement of nerve-endings. Take cancer, where one had a growing tumour, and the pain was often due to neuritis. He, too, was not sure that the cystic ovary was so painful as the cirrhotic ovary. Mr Carmichael referred to the probability of the infection being secondary to peritonitis; that was what they were driving at in their paper. In certain (three) cases it seemed to be likely that the tubercle bacillus was present in

the peritoneal fluid, and from this the tube had become infected, just in the same way as a septic condition in the tube was set up by the *Bacillus coli*. The suggestion they made was that the infection was set up in the Fallopian tube. They quite recognised, with Dr Young, that many others had already pointed out these facts.

Meeting V.—February 8, 1911.

On the evening of February 8, in place of the ordinary meeting of the Society, a complimentary dinner to Dr William Craig, on the occasion of his resignation of the office of treasurer, was given in the Caledonian Station Hotel.

About fifty Fellows and guests sat down under the chairmanship of Dr Haultain, the President of the Society. Among those present were Emeritus Professor Sir Alexander Simpson, Professor Stephenson of Aberdeen, Professor Sir J. Halliday Croom, and Dr Byrom Bramwell (President of the Royal College of Physicians). After Dr Haultain had proposed the toast of "The King," Sir Alexander R. Simpson, on behalf of the Society, presented Dr Craig with a silver tea and coffee service, as a recognition of his valuable services as treasurer for thirty-five years. He referred to the remarkable improvement in the financial condition of the Society that had been effected during Dr Craig's stewardship. During his long tenure of office he had, by his enthusiasm and financial ability, raised the Society from the verge of bankruptcy to its present flourishing condition. Dr Craig's name was indissolubly connected with the annual publication of the *Transactions*, and their free transmission to the Fellows of the Society throughout the world. They all held Dr Craig in the highest esteem, and were deeply grateful to him.

In reply, Dr Craig gave interesting reminiscences of his connection with the Society in the earlier years of its existence.

The health of the President was proposed by the President of the Royal College of Physicians, and subsequently an enjoyable programme of song and anecdote was contributed by various of the Fellows present.

MEETING VI.—MAY 10, 1911.

Dr F. W. HAULTAIN, *President, in the Chair.*

I. *The President* showed—(*a*) ŒDEMATOUS FŒTUS (Photographs). An interesting feature of the case was that, as is usual in such cases, the mother was extremely œdematous when the child was born. A peculiar point was that two days after delivery the mother passed over 120 oz. of urine and then became natural in size. Dr Haultain had never read of the discharge of urine in this way. It was of course exceedingly difficult to account for the œdema in the fœtus as well as the mother.

(*b*) SARCOMA OF UTERUS, removed from a woman, æt. 54, who had ceased menstruating a number of years previously until last November—about five months ago—when she complained of an offensive discharge from the uterus, which was associated with a very considerable amount of bleeding. The patient was a stout, plethoric woman, and her general appearance was excellent. On examination the uterus was found to be enlarged, and he decided to remove it. On removal it was found to be soft in consistency, and showed typical signs of some malignant growth. The whole uterus was found to be full of the growth, the mucosa was infiltrated throughout, and in certain parts the muscular wall had entirely disappeared. On microscopical examination it appeared to be an epithelioma; he was not prepared to say exactly what it was. He thought it was a sarcomatous condition; to call it an endothelial condition was rather a refinement. It was a very marked example of a sarcomatous tumour. What the result of the operation would be he could not say.

Dr B. P. Watson said the specimen reminded him of one which he had shown to the Society some little time ago for Dr Fordyce. At the time it was thought to be a sarcoma, but

subsequent examination showed it to be not an ordinary sarcoma but a perithelioma—one of the types of endotheliomata.

(*c*) EXTRA-UTERINE PREGNANCY, WITH PERFORATION OF UTERUS. The specimen was shown because of the history and the difficulty in diagnosis. The patient, æt. 40, had her last child in June; she nursed the child herself, and during the period of nursing—about six weeks ago—suddenly commenced to have a very severe pain in her side. Her doctor treated her in the usual way with hot applications with benefit. A fortnight later there was a very severe hæmorrhage which stopped and then recurred, and her doctor curetted her for the bleeding. This did not seem to stop the hæmorrhage, and the pain recurred; consequently Dr Haultain was asked to see the patient, and found her to have a well-marked swelling on the right side of the uterus, which gave all the appearances clinically of an extra-uterine pregnancy. The fact that the woman was nursing at the time the symptoms arose, made the diagnosis extremely difficult. The only course was to open the abdomen and remove the lump. On opening the abdomen he was surprised to find the uterus perforated. It was difficult to explain it, but seeing that had happened he decided to remove the uterus. The ovary on the other side was not healthy, so he removed it, and also the uterus along with the extra-uterine pregnancy. After the operation he learned that, when curetting the patient, the doctor had perforated the uterus. It did not seem to have done the patient any particular harm nor to have caused additional pain. An interesting point was the condition of the opposite tube, it showed no appearance of decidual reaction; this, in comparison with a previous case he had had, was interesting. The specimen showed the perforation of the uterus, but not so well as it did at the time of operation.

(*d*) MICROSCOPIC SECTIONS OF DIFFUSE ADENOMA OF VAGINA. One of the sections was from a case which he (Dr Haultain)

had himself seen, and showed in a very well-marked manner the adenomatous condition of the vagina. The other section was from the only other case recorded, namely, the case of Bonney and Berkeley, and showed the same variety of growth.

II. NOTE ON A CASE OF ADENOMA OF THE VAGINA UNDER OBSERVATION FOR FIFTEEN YEARS.

By F. W. N. Haultain, M.D.

In September 1896 I was consulted by Mrs S., æt. 35, who complained of a copious watery discharge from the vagina and something protruding from the vulva. On examination I found a swelling of the anterior vaginal wall, which protruded slightly from the vulva and simulated a cystocele. It was hard and well defined—about the size of a walnut. The vaginal walls were of an intensely pink colour, and secreted freely a thin, watery discharge.

I removed the swelling on the anterior vaginal wall, which though apparently well defined was extremely difficult to enucleate. On section it presented the appearance of a fibro-adenoma. I intended to show it at the Obstetrical Society, but, unfortunately, my museum was burned and the specimen destroyed. I at the same time removed a portion of the vaginal wall and found it covered with an adenomatous structure, which closely resembled the appearance of a cervical erosion. Subsequently I thoroughly scraped the vagina and swabbed it with tincture of iodine, in the hope that I might destroy the glandular growth.

For some months she was much better, but returned later with the same watery discharge. As the vagina presented a similar appearance as before, I repeated the curetting and swabbed with pure carbolic. This, again, cured the discharge for a period of about eighteen months, but it reappeared, and

Diffuse Adenoma of Vagina. On right, ordinary squamous epithelial covering: on left, papillary glandular covering with adenomatous infiltration of vaginal wall.

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the vaginal walls presented the same pink appearance. As she was much averse to any further operative procedure, I recommended syringing with astringents such as alum and sulphate of copper, but these exercised very little beneficial effect.

I lost sight of her entirely until June last year (1910), when she again consulted me, complaining of severe heat in the vagina and frequency of micturition. On examination I now found the vaginal walls greatly thickened, of the same pinkish colour, and showing warty-like protuberances on the surface. I again excised a small portion of the vaginal walls for diagnostic purposes, and found it to show, as the microscopic specimen demonstrates (see Plate), the same adenomatous character as previously, but very much more exaggerated, the glands now penetrating and replacing the squamous epithelial covering and dipping deeply into the muscular tissues. She was now soiling from eight to ten diapers a day with the thin, watery discharge, and said she had done so more or less for the last eight years. At no time had she complained of irregular hæmorrhage, and there was no evidence of ulceration by examination. She gradually became weaker, and died of pure asthenia on the 15th March last.

Beyond considerable discomfort from her bladder, there was no great pain suffered. Unfortunately, post-mortem examination was refused.

There can be little doubt that the case was a typical example of a diffuse adenoma of the vagina, simple in its early stages, but eventually in the course of time, from penetrating the surrounding structures, becoming what might be termed adenoma malignum.

A similar case in its early stages has been described by Bonney, and was stated by him to be unique, so that the condition must be considered of great rarity.

In these days of thoroughly surgical interference I would

not hesitate, if a similar case presented itself, to remove thoroughly the uterus and vagina, as it seems to me that, though simple in origin, all such cases must tend eventually to pursue the course which was followed in this instance. As to the origin of this extraordinary condition I am not prepared to give an opinion. Both Berry Hart and Bonney consider it to be due to some developmental anomaly associated with the Wolffian ducts. These, as is well known, are supposed to account for the formation of vaginal cysts, which, however, differ very much in the character of their epithelium from the case above described. This, as is shown in the micro-photographs, is of a cylindrical type, resembling closely the lining of the cervical glands. It is possible, therefore, that the condition may be due to an exaggerated formation of cervical erosion spreading to the vaginal walls.

III. ADENOMA VAGINÆ DIFFUSUM (ADENOMATOSIS VAGINÆ), WITH A CRITICAL DISCUSSION OF PRESENT VIEWS OF VAGINAL AND HYMENEAL DEVELOPMENT. (With Lantern Demonstration.)

By D. Berry Hart, M.D., F.R.C.P., Lecturer on Midwifery, School of Medicine of the Royal Colleges, Edinburgh.

This exceedingly rare condition has been recently described for the first time by Bowley and Bryden Glendinning.[1] Bowley and Glendinning describe the clinical features as follows:[2]— A red, granular, honeycombed appearance of the vaginal walls, small cysts, and a continuous secretion of a mucous fluid like white of egg.

[1] Bowley and Glendinning, "Adenomatosis Vaginæ, a Hitherto Undescribed Condition," *Proc. Roy. Soc. Med.*, iv.; *Obstet. and Gynæc.*, I. Trans. p. 18.

[2] A case has also been recorded by Dr Haultain, in whose practice it occurred some time ago.

T. S. genital cord, 6-7 weeks' fœtus; in front, urinogenital sinus; between sinus and pouch of Douglas, Müllerian and Wolffian ducts; behind pouch of Douglas, rectum.

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Pieces of the vaginal lining, when removed and submitted to microscopical examination, gave the following condition:— Epithelium poorly stratified, reduced to a single layer at many places; fibro-muscular stroma, with the muscular element predominant but ill-formed; tissue permeated with numerous gland-spaces lined by a single layer of columnar epithelium, which was traced in continuity with the superficial layer; no elastic tissue immediately under the epithelium, but its presence round the vessels.

In Dr Haultain's case, malignant disease developed later.

I now take up in explanation of this condition the question of the development of the vaginal tract in the human female, and also in marsupials. This involves the consideration both of vaginal development and also of the nature of the hymen. The first has special bearings on diffuse adenoma of the vagina, but I purpose at the end of the paper also taking up critically the development of the hymen.

If we look at a transverse section of the genital cord[1] in a human embryo about the sixth-seventh week we see in it three lumina; the two outer are the Wolffian ducts, separate, the mesial one the two Müllerian ducts coalesced. It is important to notice that at this early stage—sixth-seventh week—the septum between the Müllerian ducts has disappeared. The Müllerian duct is lined at this time with a low columnar epithelium. At a certain level of the urinogenital sinus corresponding to a point in the adult vagina an inch or so above the hymen there is a distinct projection on the posterior wall of the duct—the eminence of Müller. Here the Müllerian ducts end blindly. Below the eminence of Müller the Wolffian ducts open one on each side (Plate I., Fig. 1). The urinogenital sinus is relatively long at this period, and indeed has been described

[1] The genital cord is practically the part where the ducts of Müller, Wolffian ducts, and urinogenital sinus are embedded in connective tissue, and not free at any part of their periphery.

erroneously as the future vagina. At this stage, then, the Müllerian duct, which will form part of the future vagina, has a single lumen, is lined with columnar epithelium, has no lower aperture, and no hymen is present. In earlier sections the ducts are double.

The next distinct stage of development known to me is at the fourteenth week, and this is an important period, as it is at this period of fœtal development that the hymen develops. I have examined two specimens at this time, and, so far as I know, no other similar specimens have been described, even by observers who have strong views opposed to mine on the nature of the development of the hymen. Specimens at this period are best cut in the form of a central mesial slab in sagittal central section, as the paraffin blocks are somewhat large, and this direction gives a useful view of the whole tract.

In such a section one can see from above down—(1) The uterus with a lumen and beginning formation of glands; (2) the lower end of the cervical canal blocked with epithelium; (3) the vagina solid with cells, and with a line of clear space between them and the connective-tissue wall of the duct; (4) at the lower end, a remarkable condition, viz. a development of two bulbs of epithelium with cells exactly like the squamous epithelium of the adult vagina, and into one of these I traced the Wolffian duct. From these bulbs—the Wolffian bulbs—the squamous epithelium spreads up into the Müllerian duct, blocks it, absorbs its epithelium, maps out the future fornices, solid also, and passes a short distance into the cervical canal. Outside the bulbs, and higher up, the epithelial streams from each bulb communicate before entering the Müllerian ducts, and one can still see the Wolffian duct lying by the side of the vagina as would be expected (Plates II. and III.).

Thus, at or about the fourteenth week of fœtal life two lateral bulbs develop at the lower ends of the Wolffian ducts at a level a little below the eminence of Müller, where these open

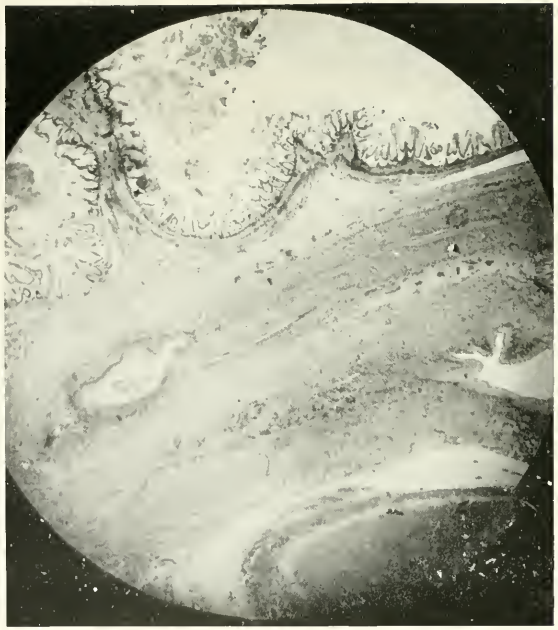

FIG. 1.

Shows sagittal mesial section of 6-7 weeks' fœtus; at left side coalesced Wolffian bulbs passing into Müllerian solid duct; below, Wolffian duct.

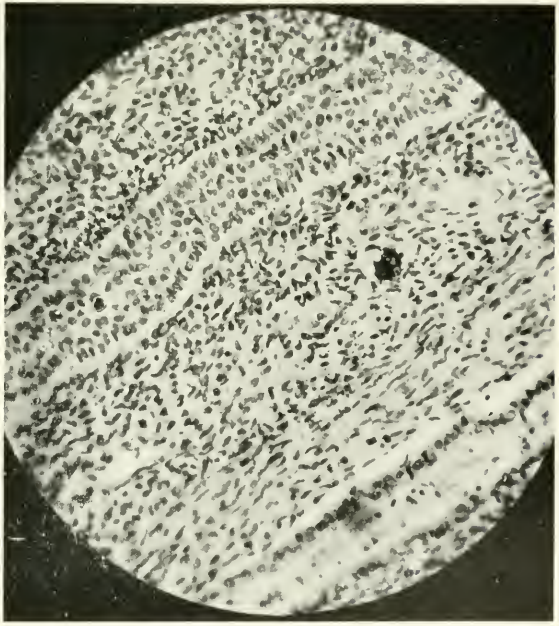

FIG. 2.

Shows solid Müllerian duct above and Wolffian duct below (14th week).

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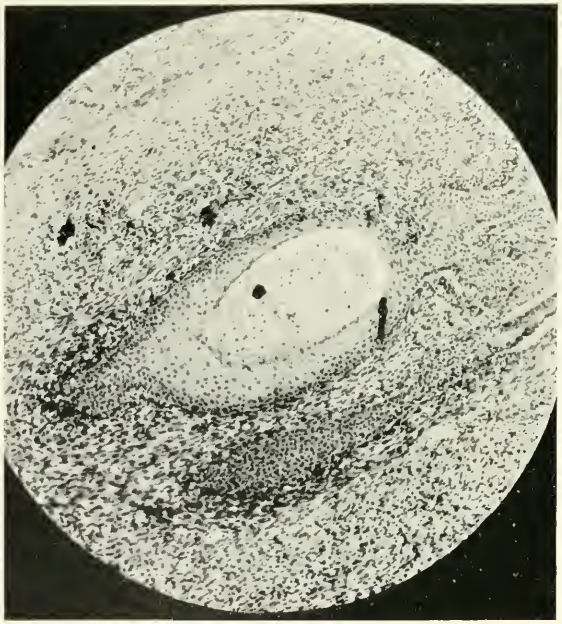

Fig. 1.

Shows large Wolffian bulb above, with epithelium passing into Müllerian duct; below is second Wolffian bulb with Wolffian duct continuous (14th week).

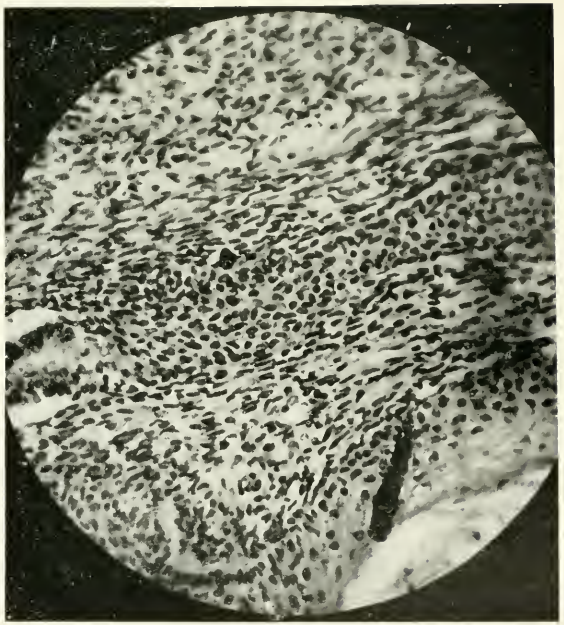

Fig. 2.

Shows Wolffian duct ending in small bulb and part of larger bulb in front at lower right corner.

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into the urinogenital sinus, and thus a little below the site where the Müllerian duct forming part of the vagina ends blindly. These bulbs proliferate in two united columns, pass up into the Müllerian vagina, absorbing its epithelium, and then this solid ingrowth breaks down centrally and gives once more a vaginal lumen. This, however, is now of a stratified squamous nature, able to stand the friction of labour. It is analogous to skin in its structure, and differs from it in not possessing hair or glands.[1]

We get a very striking illustration of this method of development of the vaginal tract if we consider the structure, naked-eye and microscopic, of the vagina in marsupials.

The Vaginal Tract in Marsupials.—The vaginal tract in the marsupials is very remarkable, as it is made up of three canals, known as the lateral canals, and the central canal. The two lateral canals communicate with the upper part of the central one, and open into the urinogenital sinus below. The central canal is a closed pouch usually, but in some, for instance in Bennett's kangaroo, the central pouch communicates directly with the urinogenital sinus. When it does not, it is said that in parturition the embryo passes from the central pouch into the urinogenital sinus, thus breaking down the separating tissue. This is a very extraordinary fact, and it may be that the other view, viz. that the fœtus passes down the lateral canals, is correct.

In 1907 I examined microscopically the entire genital tract (one lateral half) of the rat kangaroo, and found a very remarkable condition present.

The lateral canals are lined with a multiple-layered epithelium like that of the adult human vagina, while the central portion has a glandular epithelium. There is thus preserved in the central canal the temporary condition of the

[1] Von Preusschen long ago described occasional glands in the adult vagina, a fact explained by its development given above.

Müllerian ducts in the human embryo, and in the lateral canals, epithelium the same as the permanent epithelium which removes and displaces the Müllerian epithelium in the human embryo. The lateral canals are therefore Wolffian ducts in whole or in part (v. d. Broek), the central canal the Müllerian ducts combined, or cervix according to v. d. Broek.

In considering this point again, in view of v. d. Broek's criticism that the whole lateral canals are not a vaginal element, it occurred to me to look over Owen's specimens again. In one of them there is present what looks like an os externum, at or about the lower third of the lateral canal. If this be right, then below the os externum is vaginal; above it, up to the top of the central point, uterine. As it is lined with squamous epithelium in many layers, it corresponds to the part of cervical canal in the human fœtus temporarily lined with squamous cells like the vagina.

Diffuse adenoma of the vagina in the human female is thus a persistence of a fœtal condition owing to the epithelium of the Müllerian ducts remaining permanent, probably from its not having been absorbed and replaced completely by the ectodermic cells of the Wolffian bulbs.

I wish now to discuss the various views of the nature and development of the hymen.

Within the past few years the question of the development of the vagina and hymen has been a good deal investigated, but observers are still at variance in their opinions, and as I am mainly responsible for one of these, viz. that the vagina is Müllerian in its upper two-thirds and urinogenital sinus in its lowest third, and that the hymen originates from the lower ends of the Wolffian ducts and is a development in the urinogenital sinus, I purpose reopening the subject and considering critically its main points.

In the adult virgin female the relations and naked-eye anatomy of the hymen can be observed in an operative case

when the patient is under chloroform and in the lithotomy posture. If the labia minora are drawn not too far apart, it will be found to be a double pouting sagittal mesial ridge with the edges of the slit-like opening in apposition (Cullingworth), the upper coalesced ends often overlapping the urethral orifice. The alleged various forms of normal hymen, where the aperture is described as oval, crescentic, round, and so on, are due to lateral traction on the edges of the hymen, causing disturbance in their normal apposition. Even when the hymen has a double slit the above applies.

On naked-eye examination of the hymen in anatomical virgin specimens, one can see on its inner aspect rugæ continuous with those of the vagina, as Budin pointed out. Its covering is more analogous to skin, a fact often rightly emphasised as regards the vagina. The microscopical structure of the hymen has been well described by Gellhorn among others. It is covered with a many-layered epithelium on papillæ of connective tissue, with elastic and sometimes unstriped muscle in its substance; special nerve-endings have been observed. In the hymen in some atresic cases it has been noted that the inner surface is covered with single-layered epithelium, the outer with multiple-layered epithelium.

Nagel, in a sagittal section of a 4 cm. fœtus, describes the eminence of Müller as made up of special protoplasmic cells, and considers the cells at the level of that part of the Müllerian ducts forming the future vagina to be of a different nature from those at a higher level. Any change in the epithelium afterwards he considers as a local development and not an invasion from any other source.

It is very generally held that the hymen is the thinned-out lower end of the vaginal walls. Budin, who stated this view very clearly and fairly, studied it from the point of view of naked-eye anatomy, and pointed out that the vaginal rugæ could be traced on the inner surface of the hymen. In this he

is quite correct, but in a conversation that I had with him on the subject he stated that this was not conclusive evidence that the hymen had not an origin from the Wolffian ducts.

Pozzi and others have associated the hymen with the vulva, and Matthews Duncan drew attention to the fact that in ordinary atresia hymenealis leading to the retention of menstrual blood the edge of the hymen was present. This I have verified in some cases, and found that the hymeneal edge fringed the occluding membrane.

The view that the hymen is vulvar[1] in its nature is quite untenable. The hymen lies, in the adult virgin, in the lower part of the former urinogenital sinus. It is separated from the labia minora, has the vestibule above it, and the vaginal aspect of the fossa navicularis between it and the fourchette. Those who hold the view argue by imperfect exclusion, and conclude that if the hymen is not Müllerian it must belong to the external genitals, thus leaving the urinogenital sinus quite out of consideration.

An important contribution to the development of the vagina was made by Tourneux and Legay, and by Nagel, and confirmed by myself and others, when it was pointed out that at one stage of its development the Müllerian lumen became blocked by epithelial cells and was therefore solid for a time.

Klein and Nagel state that at about the fourth and a half month the lower end of the vagina is thus blocked by a mass of epithelial cells, and Klein considered that it was by this breaking down and by an involution from below that the hymeneal aperture was made.

According to Nagel, "the Müllerian ducts are originally composed of large protoplasmic cells of an epithelial character. Around this mass of cells, which gradually becomes a tube, the mesodermal formative cells are grouped in a circular arrangement, thus forming the first Anlage of the connective tissue

[1] This view does not include the urinogenital sinus in the vulva.

and muscular layer of the Müllerian ducts; in other words, the connective tissue appears after the ducts are established, and grows in proportion to and together with the downward growth of the ducts. Thus when the ends of the ducts protrude into the sinus in the form of an epithelial protuberance, their connective tissue comes down with them, grows into the terminal conus, and forms the inner or central layer of the protuberance."

If this be the true mode of origin, "the hymen" (Gellhorn urges) "is a product of the Müllerian ducts exclusively. The epithelium of the sinus urinogenitalis does not play any active part but only covers the outer layers of the epithelium of the Müllerian ducts in the form of a very thin cuticle. Thus it is readily understood why the epithelium of the vulva can be dissected from the hymen as recorded above in connection with the theories of Pozzi and Schaffer" (Gellhorn, *op. cit.*, p. 495). Nagel objects to my view that the Wolffian ducts I described may be Bartholinian or a low implanted ureter. The Bartholinian duct, however, has not developed at this period, but does so about the fourth and a half month, and its axis is different, seeing that it runs transversely in to end on the outer aspect of the hymen. A low implanted ureter is exceedingly rare in the adult, and only a few cases have been recorded. I have described such, and shown that the so-called implantation of the ureter is due to a persistent communication of Wolffian duct and ureter; and when the normal development of the ureter and part of the kidney is considered, viz. that it is due to a budding upwards from the Wolffian duct to meet the part developed above from mesoblast, the cause of the anomaly is at once evident.

J. C. Webster of Chicago, in a probationary essay on the "Early Wolffian and Müllerian Ducts" for the Fellowship of the American Gynæcological Society, opposes my views and agrees with those of Nagel. The most noteworthy feature of

Webster's paper is that he describes no specimens bearing on the development of the hymen. His paper is thus a purely literary one, and not of scientific importance. He is not consistent in his criticism, as at p. 457 he states—"After the careful researches of Nagel and Hart regarding the origin of the hymen, which I have recently corroborated . . ."—but from the fact that he describes or figures no specimens and had previously disagreed with me, the position he there takes up is a puzzling one.

Gellhorn in his paper urges against my view that "systematic researches of other investigators result in an altogether different conception of the course of the Wolffian ducts. Until recently the majority of observers believed that the Wolffian ducts terminated at or near the cervix uteri in the upper part of the vagina. This view can no longer be held. Beigel, Dohrn, v. Ackeren, and others first described *rests* of the Wolffian ducts along the entire length of the vagina, and Klein, in 1897, demonstrated in serial sections the course of the Wolffian ducts within the hymen. It must be noted that v. Ackeren, Klein, and Groschuff, Seitz, Hengge, the last three working with Klein, found the Wolffian ducts within the hymen" (Gellhorn, *op. cit.*, pp. 428, 429). These facts, however, are not only not against my view but are directly confirmatory of it. Had the Wolffian ducts normally ended above the level of the hymen, that might have been fatal to it. What Gellhorn finds in the quotation given against my view I fail entirely to see; in fact it is totally irrelevant, and I see no point in the quotation hostile to my view at all.

Kempe has shown that in the white rat the hymen develops from a bridge of tissue below the ends of the Müllerian ducts (see Quain's *Embryology*, T. H. Bryce).

Dr Wood Jones, in a valuable paper, has been the first observer to confirm the existence of the double bulbs I have described, and he considers them to be derived from the

Müllerian ducts, but the details are not given and there is thus no opportunity of discussing his views. He believes the hymen to be "the tissue these bulbs do not penetrate." He also confirms my statement as to the urethra becoming occluded by the bulbs and re-tunnelled, a process analogous to what happens at the vaginal orifice. Indeed some observers have described what they term a urethral hymen, which is probably due to this urethral perforation.

One difficulty in accepting Dr Wood Jones's view is that even at the sixth week the Müllerian duct in the genital cord has a lumen and is single. Dr Jones's view, apart from other points, would require the Müllerian ducts to re-acquire a double lumen.

The view that the hymen is Müllerian has at first sight much to recommend it, but it fails to account for several facts. In Nagel's account he figures, as I have already noted, in a 4 cm. fœtus the urinogenital sinus with the Müllerian eminence made up of well-marked protoplasmic cells, the Anlage of the hymen and vagina, and considers the cells higher up where the uterus and tubes form to be different. The ultimate vaginal lining he considers to be due to a local proliferation, and he figures this is as blocking the vagina in a three months' fœtus. Those who accept Nagel's view are omitting the consideration of the all-important stage of the double bulbs I was the first to note. All other observers who figure the epithelium blocking the vagina figure it as a single and local epithelial plug. Of course it is a very evident deduction from their facts that this proliferation and distension open up the lower end of the vagina and form the hymen. These two far apart stages of Nagel from a 4 cm. fœtus to one at the third and a half month are not the only ones, and to criticise my view on them alone must lead to an erroneous conclusion. Gellhorn, indeed, takes this one stage of Nagel's in the 4 cm. embryo, and writes as follows:—"From this eminence the

hymen is formed. The fine details may to my mind be conceived as follows:—When the united Müllerian ducts reach the sinus urinogenitalis they push the thin layer of epithelium lining the sinus forward, and by so doing they make this epithelium even thinner than before. This condition is clearly seen in Nagel's picture. In this stage the lower ends of the Müllerian ducts represent a somewhat cone-shaped solid epithelial cord. The formation of a lumen proceeds from above downwards, and this canalisation corresponds with the course of the ducts themselves, with its concavity anterior. Therefore the point where the lumen of the duct breaks through the solid end is as a rule nearer the upper border of the conus and so is responsible for the production of the semi-lunar form of the hymen, which is by far the most common. The concave course of the Müllerian ducts is due to the curved abdomen of the embryo. The extremity of the conus (Müllerian eminence), with a more or less eccentric lumen projecting into the sinus, consists at first only of the epithelium, covered on the outer surface with a very thin layer of sinus epithelium. Only secondarily is this mass of epithelium invaded by connective tissue" (Gellhorn, pp. 434, 435).

Gellhorn admits that this account of his as to the orgin is almost entirely hypothetical, and it is indeed remarkable as a piece of induction. He describes the development as occurring in the stage of the 4 cm. fœtus of Nagel, and does not figure any subsequent stage or indeed describe any such until the fourth month of Nagel's specimens. The hymen is not formed at this early stage but at a much later one—three to four months—so that he rejects my views on insufficient grounds and brings forward a purely hypothetical and wildly improbable theory.

Taussig agrees with Webster and states (in a footnote) that "Webster has fully pointed out the fallacies of his (Hart's) conclusions." He further states that he has assumed that the

vagina is entirely formed by the coalesced Müllerian ducts and not to any extent by the urinogenital sinus (p. 99), *i.e.*, he assumes what he should prove or disprove, and rejects actual specimens in favour of a mistaken preconception.

Bolk in a recent important paper gives the history well, and his conclusions are as follows:—

"Nach Dohrn entwickelt sich somit die Klappe sekundär, hat nichts mit der ursprünglichen Müller'schen Papille zu tun und ist die hymenale Offnung eine primäre Offnung, nämlich das nicht ganz verschlosene Lumen des Scheidenkanales. Doch bleibt immerhin der Hymen eine vaginale Bildung. Eine dritte Ansicht, die hier kurz erwähnt zu werden verdient, ist jene von Berry Hart. Die morphologische Deutung der Scheidenklappe, die dieser Autor gibt, hängt mit seiner Auffassung der Entstehung der Vagina zusammen. Es sollte nämlich der untere Dritteil der Scheide nicht von den Müller'schen, sondern von den Wolff'schen Gängen gebildet worden sein und demzufolge musz der Hymen als ein Produkt der terminalen Enden dieser Kanäle angeschen werden; so sagt der Autor (l. c. S. 342): 'thus the hymen is formed by a special bulbous development of the lower ends of the two Wolffian ducts, aided by an epithelial involution from below of the cells lining the urogenital sinus.' Schlieszlich musz die Auffassung von Pozzi erwähnt warden, der den Hymen als eine vulväre Formation deutet, entstanden aus zwei seitlichen Falten, die sich auf die Seitenwände des primitiven Sinus urogenitalis bilden, und einander entgegenwachsend in der Medianlinie sich verbinden.[1] Der Hymen würde somit paariger Herkunft sein und von dem Müller'schen Hügel ganz unabhängig sein.

"Der zweite Punkt betrifft die Herkunft der Vagina. Die

[1] Pozzi's view has been classed with those deriving from the external genitals, omitting the fact that he places the urinogenital sinus with them.

älteren Autoren (Müller, Valentin, Rathke) meinten, dass die Scheide eine differenzierter Teil des Sinus urogenitalis war, eine Ansicht, die durch spätere Untersuchungen als nicht richtig erkannt worden ist. Weitaus die meisten Autoren erblicken jetzt in der Vagina ein Bildungsprodukt ausschlieszlich der Müller'schen Gänge. Nur einige wenige Autoren nehmen einen etwas anderen Standpunkt ein. Jener von Berry Hart geht schon aus dem Obengesagten hervor, und wird weiter deutlich durch die zwei folgenden Sätze: 'The upper two-thirds of the vagina are derived from the ducts of Müller, and the lower third is due to the coalescence of the upper portion of the urogenital sinus and the lower ends of the Wolffian ducts' (i. c. S. 344). Auch Kempe hat sich, was die weisze Ratte betrifft, für eine Beteiligung der Urnierengänge an der Bildung der Vagina ausgesprochen" (Bolk, pp. 252, 253).

Bolk's final conclusions are:—

"Kehren wir nach diesem Exeurs zu unserem Gegenstand zurück. Auf Grund meiner Untersuchungen bin ich somit bezüglich der Entwicklung der Urethra, des Septum urogenitale, der Vagina und des Hymen zu einer Auffassung gelangt, die von der mehr geläufigen wesentlich abweicht. In den folgenden Sätzen findet man kurz zusammengefasst die Ergebnisse meiner Untersuchung übersichtlich dargestellt:—

"1. Die Urethra des Menschen ist doppelter Herkunft; der obere Teil geht aus der gemeinschaftlichen Blasen-Urethral-Anlage hervor, der untere Teil stammt vom primitiven Sinus urogenitalis her.

"2. Der kaudale Abschnitt des Septum urogenitale beim Menschen entsteht durch ein Zusammenwachsen zweier Falten, die beiderseitig von der Seitenwand des primitiven Sinus urogenitalis sich erheben, einander nähern, in der Medianebene zur Verschmelzung kommen und in dieser Weise das primitive Septum nach unten verlängern.

"3. Die Vagina des Menschen ist doppelter Herkunft; zum

grössten Teil geht sie aus dem untern Abschnitt der verschmolzenen Müller'schen Gänge hervor, dieser Abschnitt ist als 'Pars-Müllerica' zu unterscheiden; der untere kleinere Teil (vielleicht etwas das untere Drittel) lässt sich vom primitiven Sinus urogenitalis ableiten, diesen Teil möchte ich 'Pars adjuncta' nennen.

"4. Der Hymen ist eine durch Faltenbildung der Seitenwände des primitiven Sinus urogenitalis enstandene Klappe, die paariger Herkunft ist, durch Übergreifen auf die Hinterwand des primitiven Sinus urogenitalis sich zu einer halbmondförmigen Membran ausbildet und durch eine Verschmelzung auch der vorderen Enden beider Falten sich zu einer mehr ringförmigen Klappe entwickelt.

"5. Das Orificium hymenale ist einer primäre Offnung.

"6. Das Vestibulum vaginæ stellt nur einen Teil des primitiven Sinus urogenitalis dar.

"7. Das Perineum anterius (Carina urethralis) ist entwicklungs geschichtlich progressiver Natur" (pp. 265, 266).

My own views, confirmed by Bolk, as stated in 1901, are as follows (*Edin. Obstet. Trans.*, 1900-1, xxvi., 273):—

1. The upper two-thirds of the vagina are derived from the ducts of Müller.

2. The lower third is due to the coalescence of the upper portion of the urinogenital sinus and the lower ends of the Wolffian ducts.

3. The epithelial lining of the vagina is derived from the Wolffian bulbs, which again are epithelial proliferations of the lower ends of the Wolffian ducts. The vaginal mucous membrane is thus ectodermic.

The hymen forms at the Wolffian-ducts-opening, and not at the eminence of Müller as usually stated. I think it probable that the Wolffian bulbs block the urinogenital sinus, and that the lumen is re-established as a double one, urethral and vaginal, by an epithelial central resolution and by involutions

from the sinus below. Thus in the adult female the vestibule, lower third of the vagina, and lower end of the urethra are derived from the sinus urinogenitalis (pp. 273, 289) (Plates IV. and V.).

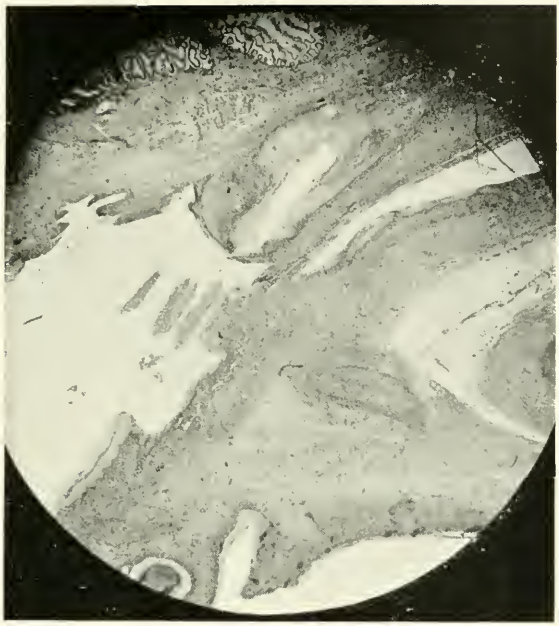
In the new edition of Veit's *Handbuch der Gynäkologie*, Spuler in a comprehensive article emphasises the above views (Bolk's and my own), and gives also his own researches on the comparative anatomy of this region in cavia—felis, ovis, sus—specially on the cavy.

The following are his conclusions (1908):—

"Bei meinen Untersuchungen kam ich in einem wesentlichen Punkt zu einem von den Bolkschen abweichenden Resultat. Im November 1907 habe ich in der Erlanger Societas physico-medica mitgeteilt: Die Vagina entsteht nach Untersuchungen an Cavia, Felis, Ovis, Sus, speziell an Cavia, und auch an menschlichen Embryonen, aus zweierlei Bildungen; aus einem in den Müllerschen Hügel einwachsenden, dem Epithel des Sinus urogenitalis enstammenden 'Conus vaginalis,' der sich später beim Menschen auszerordentlich stark entwickelt und den grösszten Teil der Scheide liefert, und durch Aufteilung des grösszeren Teiles des Sinus urogenitalis durch zwei ungefähr frontal gestellte Falten in einen vorderen Kanal die sekundäre weibliche Harnröhre, und einen hinteren, den unteren Teil der Scheide; nur der kleine ungeteilt bleibende Bezirk des Sinus urogenitalis wird zum Vestibulum vaginæ" (Spuler, Veit, v. pp. 620-621).

We may thus consider the view of the entire Müllerian origin of the vagina and hymen as overturned and its origin in part (upper two-thirds) from Müller's ducts, in part from the upper portion of the urinogenital sinus, as established. Thus the hymen is not an organ of the Müllerian ducts nor of the vulva, but of the urinogenital sinus.

The question of vaginal atresia, and especially of atresia hymenealis, is intimately bound up with this whole question.



Formation of hymen. To left is urinogenital sinus (vestibule), and above Wolffian bulbs breaking down.

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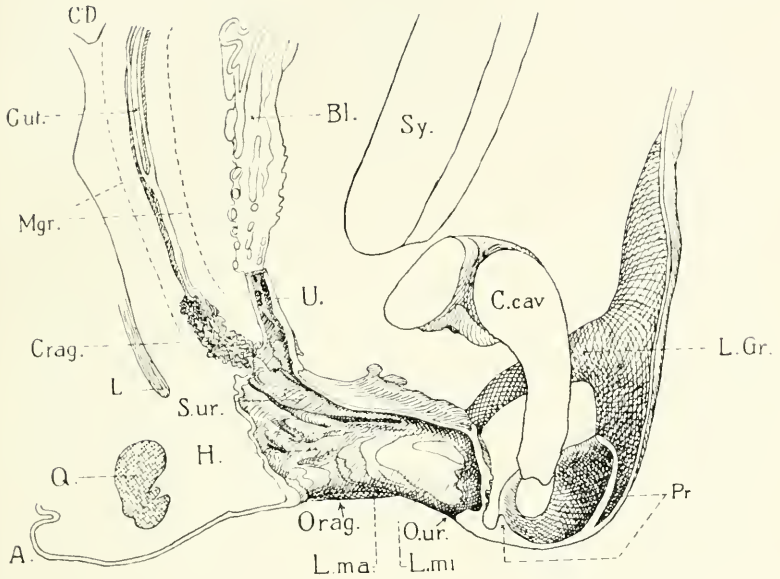

Mesial sections through the urinogenital sinus of a 13·5 mm. (head-breech length) female embryo, viewed from within. A., Anterior end of anus; Q. M., Sphincter ani; L., Longitudinal muscle of rectum; C. D., Lower end of pouch of Douglas, posterior; C. ut., Cavum uteri; M. gr., Boundary of uterine mesenchyme; C. rag. (instead of vag.), Conus vaginalis; Bl., Bladder; U., Urethra; S. ur. (instead of uv.), Plica septi uro-vaginalis; O. ur., Anlage of the ostium urethræ; Sy., Symphysis; C. cav., Corpus cavernosum clitoridis; Pr., Prepuce fold; L. gr., Boundary opposite labium majus; L. ma., Labium majus; L. mi., Anlage of the labium minus (Spuler, *op. cit.*, fig. 35, p. 628).

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One special point emerges, viz. that in atresia the initial defect is that the non-breaking down of the solid epithelial formation in the vagina and cervix is the central fact to be taken into account in the formation of many of them, and that when this gives a feasible explanation, as it does in most forms, hypothetical inflammatory changes need not be considered. In atresia hymenalis I have always seen the edges of the hymen and an unbroken diaphragm more or less thick between them. Some observers allege that in such the hymen may not be present. It has always seemed to me that the preponderating number of cases, at any rate in which the hymen edges are present, is due to the fact that the development of the hymen is intimately bound up with that of the ducts of such important early secreting organs as the Wolffian ones, organs essential to healthy development, and therefore structures whose absence or malformation entails serious nutritional defects. When the blocking epithelium in the vagina does not break down we get varieties of atresia, and these are easily understood in the light of the development given. Thus when the epithelial plug of the two Wolffian bulbs forming the hymen does not break down we get atresia hymenealis. Very rarely is there a transverse septum an inch from the septum hymen, but I have seen two such, and it is probably the lower end of the true Müllerian vagina, where the eminence of Müller was, that is imperforate. When all the solid epithelium in the vagina does not break down we get complete atresia of the vagina; when only that in the cervix, we get a retention of blood in the uterus hæmatometra.

In review, then, it will be seen that these two views of the origin of the hymen, viz. from the Wolffian ducts and sinus or from the Müllerian ducts, are sharply contrasted as to the origin of the hymeneal and vaginal lining; the one urges a local change from early protoplasmic cells to squamous multiple epithelium, the other an invasion of squamous cells

from the Wolffian bulbs into the Müllerian duct as far as the lower third or so of the cervical canal.

The view I bring forward is an interpretation so far as possible of what appears to take place during this development, and an attempt to explain how the adult tract is formed. Development proceeds from the stage of the double Wolffian and Müllerian ducts and the urinogenital sinus with its single structure, from the blind ending of the Müllerian and patency of the Wolffian ducts, up to the formation of the adult tract, where we find the hymen present, the vagina provided with a skin-like multiple-layered lining, and uterus and tubes with a mucous membrane of a totally different nature. The Müllerian view states that the double Müllerian tubes (vaginal portions) coalesce, the epithelial lining of the hymeneal and vaginal Anlagen becomes converted into a multiple-layered epithelium which first occludes the vagina and then breaks down to form a lumen. A special increase of this epithelium at the lower end of the vagina by its bulging and pressure forms the hymen and hymeneal aperture.

How in the view of the Wolffian origin of the hymen does the vagina get its different lining as compared with the uterus and tubes? By the development of the Wolffian bulbs from an ectodermic source, viz. the lower ends of the Wolffian ducts, their coalescence to form the hymeneal ridges, and the passage of their epithelium into the vaginal tract rendering it solid up as far as the lower third of the cervical canal. If the vagina at this stage of occlusion is examined it will be seen to be, not a mere local proliferation of the epithelium lining its walls, but an actual solid plugging continuous with the epithelium of one of the bulbs. It may be urged that this description of the invasion of the vaginal lumen from the bulbs is an unusual phenomenon, and therefore more likely to be an erroneous interpretation, but it is really quite analogous to what happens in the development of other organs; thus the glans penis is

tunnelled in the same way, the open urethral canal in the male is closed in the same way, and, to take a simple instance, a sweat gland is first a solid epidermic plug with surface cells central, and develops its lumen by these breaking down. In fact, the special value of an epidermic plug as a solid structure, or in the epidermic invasion of a lumen, is that it has in its centre surface-cells with a power of desquamation thus forming the lumen. In the bulbs one sees that the central cells, which are analogous to the superficial cells of epidermis or vagina, are desquamating to form the hymeneal opening. What happens there, occurs higher up in the vagina in the vaginal fornices, and in the cervical canal in its lowest third, so that the whole genital tract is ultimately pervious and provided in each segment with a lining appropriate to its functions.

From all this it follows, as already said, that adenoma vaginæ diffusum is a persistence of an embryological condition due to a practical absence of the epithelium relining the vagina at the third month. It is of great interest to note that malignant disease supervened in Haultain's case.

The question arises as to whether anything can be done for such cases. The only thing I can suggest is either entire excision, or that flaps of skin be taken from the labia majora, and by them, after curetting, and in a way I need not detail, a skin-covering provided for the vaginal walls.

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For full literature, see Spuler and Gellhorn.

The President said he was sure they were all very much indebted to Dr Hart for giving them this interesting demonstration of a subject which he had very peculiarly made his own. The question of the epithelium of the vagina, vaginal cysts, and so on had always been a moot point. The cysts were thought to be derived from the Wolffian ducts. Cases of adenoma were perhaps not so rare as they were thought to be. Dr Hart's paper had thrown light upon a peculiar condition of adenomatous tissue in a structure where there were no glands present. They would be glad to hear any remarks; personally he was unable to speak on the subject. He would like again to thank Dr Hart for bringing the subject before them.

Dr Berry Hart said he was sorry Dr Haultain had not shown his case sooner.

The President said the interesting point about his case was the fact that he had followed it for so many years. There could be no doubt that the tendency of all these growths was towards malignancy. They were pathological disturbances and might go on for many years, fifteen years in his case—but they might be much more rapid.

IV. SHORT NOTE OF A CASE OF SUPERFŒTATION.

By Dr A. E. Turnbull, Broadway, Dorset. (Communicated by the Secretary.)

An authentic case of superfœtation is not so common as to make the following notes uninteresting. On 11th March of this year I attended a lady, æt. 24, in her first confinement; according to her reckoning she was only about six and a half months pregnant, but she was not at all certain on this point. When I first saw her at 5 p.m. on 11th March, the nurse in attendance told me that labour had begun at 3 a.m. the same day and the membranes had ruptured about an hour later.

On examining the abdomen I found the uterus extending to the xiphoid cartilage, tender on pressure, and obviously over-distended; palpation made me doubt the nurse's statement that the membranes had ruptured, as there seemed to be an excess of liquor amnii. I did not auscultate.

On vaginal examination I found the os nearly fully dilated, a head presenting, and the membranes undoubtedly ruptured; a diagnosis of twins was then made, and I put the large size of the uterus down to an excess of liquor amnii with the second twin.

Owing to the over-distension of the uterus the pains were quite ineffectual, and I delivered the first child with forceps; the membranes of the second child were then ruptured, a very large quantity of liquor amnii escaping, and a little later the

child was born. There was a single large placenta with a single chorion and two amnion. As the membranes separated and came away another body also came out of the uterus, and on examination, this proved to be a third ovum, quite complete and healthy, containing a fœtus of about three and a half months. While I was examining this third ovum the condition of the mother suddenly required my attention, and as the nurse thought I was finished with it she destroyed it with the placenta and membranes.

The twins lived only a few hours and the mother made an uneventful recovery. The age of the twins was uncertain as the mother was quite unable to give accurately the probable duration of her pregnancy, but they certainly were not older than six to six and a half months. From my examination of the third ovum I came to the conclusion that it was not more than three months old. The above case, then, is one of superfœtation; there was first a uniovular twin pregnancy with hydramnios of one sac, then there was fertilisation of the second ovum evidently about three months after the first.

This case must prove that ovulation continues at any rate during the early months of pregnancy; and, of course, there is no reason why, if ovulation continues fertilisation of a second ovum should not result so long as the decidual space remains, that is, up to above the end of the fourth month.

Dr Berry Hart said he did not think Dr Turnbull had proved his case to be one of superfœtation at all. Twins varied in size. He had just been looking into a very interesting monograph by a Dutchman, in which he described a case where the mother gave birth to five children. In that case there were five fœtuses, five placentæ, five cauls—triplets and twins.

Dr James Ritchie said he had been reading a French journal

last week, where what seemed to be an undoubted case of superfœtation was described. A woman, æt. 40, six months pregnant, was taken into hospital with copious hæmorrhage. There was discharged what appeared to be a complete ovum, which contained a three-months' macerated fœtus. Another very much smaller ovum was found attached to the uterine wall, which might have been the product of a month's conception. That could not have been a double uterus because of the single ovum, and it looked as if the case had really been one of superfœtation in a normal uterus.

Dr Berry Hart said a curious point was that one could get one placenta big and another very small; the amount of placenta made a difference in the size of the fœtus.

Dr James Ritchie said one might get a dead twin comparatively early, but in the case he had mentioned there was one very small ovum and the other was nearly a three-months' fœtus.

Dr B. P. Watson said without thorough examination Dr Turnbull could not exclude the two being of exactly the same age, one twin being dead. The dead fœtus remained in a comparatively fresh condition, and might look as if it had only died a few hours before delivery.

Dr Keppie Paterson remembered being called to a case in the Canongate where the students and nurses could not deliver the child. The woman had gone to full term. She passed a three-months' pregnancy, which might have been quite fresh. Professor Simpson saw the specimen and showed it to the students. At three months it had died, and had been retained quite fresh for six months in the uterus.

Meeting VII.—June 14, 1911.

Dr F. W. N. Haultain, *President, in the Chair.*

1. *Professor J. A. C. Kynoch* showed—(*a*) ectopic gestation, showing tubal abortion in progress, removed from a patient, æt. 28, from whom a left-sided ovarian tumour had been removed a year previously. She had all the signs and symptoms of an ectopic pregnancy.

(*b*) Ectopic gestation repeated in same patient. The case was very much similar to the previous specimen. In both cases there was very little blood in the pouch of Douglas. The fact that four years previously a right-sided tubal gestation was removed was interesting; this was followed by two normal pregnancies and subsequently this typical extra-uterine gestation. His experience of repeated tubal gestation corresponded with what had been found in the majority of tabulated cases. Out of 23 cases on which he had operated, he had found that three ectopic gestations had repeated.

(*c*) Fibromyoma of cervix, removed from a patient, æt. 28, who complained of discomfort in the pelvis, but had no urgent symptoms.

(*d*) Fibromyoma of ovary, showing cystic degeneration, removed from a patient, æt. 46.

(*e*) Fibromyoma of uterus, showing red degeneration, removed from a patient, æt. 36. The uterus was removed on account of excessive hæmorrhage. There had never been any special pain, and no indications of a tumour previous to the operation. He understood that this form of red degeneration had been frequently noticed after pregnancy; in this case that was not so.

Professor Kynoch asked permission to show also:—

(*f*) Typical ovarian cyst, showing torsion of the

PEDICLE, WITH HÆMORRHAGE INTO THE CYST, which before operation suggested the possibility of ectopic gestation.

The President asked Professor Kynoch if he was sure that the specimen he had shown was a fibromyoma of the ovary?

Prof. Kynoch said the other half had been examined microscopically, and it had proved to be a fibromyoma. The patient had very few symptoms, but nothing would satisfy her till she got the swelling removed.

II. *Dr Haig Ferguson* said with the approval of the Secretary he had decided to leave two of his specimens over till the next meeting, when he would show them along with some lantern slides, and now showed:—

(*a*) BILATERAL PAPILLOMATOUS TUMOUR OF THE OVARY, removed from a patient, æt. 50. The specimen, which had shrunk considerably, was interesting from the fact that the only symptom present was distension of the abdomen by ascitic fluid. On re-accumulation of the fluid the abdomen was opened, and the tumour found.

Dr Haig Ferguson also asked permission to show:—

(*b*) A specimen he had brought which showed the treatment of the umbilical cord with rectified spirit. After the child is washed and dried, the stump of the cord is rolled out, and wrapped up in gauze which has been soaked in 90 per cent. rectified spirit. It is left undisturbed for forty-eight hours, and is then cut off flush with the skin.

III. *Dr B. P. Watson* showed—(*a*) LIP OF CERVIX GREATLY HYPERTROPHIED AND IN A STATE OF GANGRENE, removed on the third day of the puerperium, from a patient who had had four children previously. Nothing had protruded from the vagina previous to or during pregnancy. The child was delivered with forceps, without any difficulty. The doctor noticed this tag at

the time. There was considerable discharge. Dr Fordyce saw the patient, and on examining her under an anæsthetic found this tag hanging down from the anterior lip. Microscopically it showed fibrous tissue quite gangrenous at the tip. On section, the vessels at the base were thrombosed.

(*b*) UTERUS CONTAINING SEVERAL FIBROID TUMOURS AND A FOUR AND A HALF MONTHS' OVUM, removed by supravaginal hysterectomy from a patient, æt. 25, who had had an early miscarriage about ten years previously, and who had not been pregnant since. For four months there had been amenorrhœa, and the patient had noticed a swelling in the abdomen. There was a considerable amount of pain, especially over the right side of the abdomen. Her doctor found she had a bilateral ovarian tumour. Under an anæsthetic, softening of the cervix and body of the uterus could be made out on the right side; on the left side there was this big mass extending up almost under the costal margin. Owing to the pain and tenderness over the tumour, and to the fact that the patient stayed in a very inaccessible district, it was thought best to remove the uterus and tumours right away, fearing that there might be some red degeneration going on. A supravaginal hysterectomy was performed, and the patient did very well.

(*c*) UTERINE FIBROID UNDERGOING SARCOMATOUS (?) DEGENERATION (shown for Dr Berry Hart), removed by supravaginal hysterectomy by Dr Berry Hart. The interest of the specimen lay in this great swollen mass behind the upper aspect of the tumour, which at one part burst through the uterus. On microscopic examination, the appearance of the sections showed changes very like sarcomatous degeneration. The tissue was very cellular in certain parts. A lining of epithelium and large spindle cells mixed with other cells could be seen. It could not definitely be said to be sarcomatous, but the appearances suggested that. The tumour was only in a "suspicious" stage.

Dr Berry Hart, referring to Dr Watson's specimen (*a*), said some years ago he showed a case in which the labour was spontaneous. The cervix had been torn, and then a tag about the size of the little finger came down, which he removed after labour.

Dr M'Gibbon, referring to Dr Watson's specimen (*a*), asked if the gangrene had set in after labour.

Dr Watson said it had.

Dr M'Gibbon said it was such a rare condition that one wondered if there was anything to account for it.

IV. NOTE ON A CHYLOUS MESENTERIC CYST SIMULATING A PELVIC TUMOUR.

By John A. C. Kynoch, M.B., F.R.C.S.E., Professor of Obstetrics and Gynæcology, University of St Andrews; Gynæcologist, Dundee Royal Infirmary.

Mesenteric cysts are evidently among surgical rarities. Isolated cases are to be found in surgical literature, but the standard text-books which I have consulted either omit the subject altogether, or merely refer to it briefly. Although it is of surgical, rather than of gynæcological interest, the condition has been so frequently mistaken for pelvic tumours, that on this account as well as my having had the experience of a case, I have ventured to bring a note of it before the Society.

Between the layers of the mesentery we find connective tissue, fat, lymphatic vessels and glands, unstripped muscular fibre, as well as congenital remnants of the Müllerian and Wolffian ducts—all of which may give rise to tumours in this region. Of those tumours the cystic swellings may be (1) serous, (2) chylous, (3) hydatid, (4) blood cysts, (5) dermoid, (6) cystic sarcoma. Further, cystic tumours arising from the ovary may become mesenteric by later extension. Of those,

the one to which this short note refers is of the *chylous type*—a condition first carefully described by Hahn of Berlin.

The origin of those chylous cysts may be manifold. They have been described as arising from dilated lacteals, or degenerated lymph glands, and they may be unilocular or multilocular, sessile or pedunculated. Rasch reported a case before the Obstetrical Society of London in 1887, which he stated was the first recorded case of a chylous cyst in a woman. They are said to be more common in women than in men, and may be met with at any age. They vary in size. They may be so small as to be of no importance, or so large as to cause death by their bulk. The most important *physical signs* are three.

(1) The most prominent part of the tumour is near the umbilicus.

(2) Extreme mobility, so that the cyst may be readily pushed from one side of the abdomen to the other, and to a certain extent upwards and downwards.

(3) To precussion there is a note of resonance all round the tumour, with very frequently a resonant band crossing the swelling.

Clinically the most outstanding symptoms are *pain* over the tumour, *intestinal disturbances*, constipation being the rule, but there may be attacks of vomiting with persistent diarrhœa and lastly, *general wasting*—due in all probability to interference with the lacteals of the mesentery.

The case which I have to report is that of a woman 37 years of age. She was admitted to the gynæcological ward of the Dundee Infirmary, 17th April. She complained of a *pain* in the left lower part of the abdomen, where she had noticed a swelling gradually increasing in size for three years. She stated that the pain was worse on exertion, and during menstruation, which had always been quite regular. She had been married seven and a half years, and was the mother of

three children. Her labours were uncomplicated and she had had no miscarriages. She was of a healthy appearance, and except for occasional attacks of diarrhœa and vomiting her general health was good.

On abdominal examination the abdominal wall was found well developed, and in the hypogastric region there was felt a well-defined *tense tumour*, nodulated upon its surface, and extending from the left iliac fossa across the middle line for about 1 inch to the right side of the umbilicus. The mobility of the tumour was very marked, especially from side to side. There was dulness on percussion all over the tumour, with a resonant note surrounding two-thirds of the swelling.

On examination *per vaginam* a cystic swelling the size of a lemon was felt in the right fornix. The left ovary was not palpable and the uterus was somewhat enlarged. On pressing the tumour downwards towards the pelvis it appeared to be intimately connected with the uterus, which led me to take it for a left ovarian tumour or a subserous fibroid. On opening the abdomen an ordinary right-sided multilocular ovarian cyst was removed. The uterus and left ovary were normal. The abdominal tumour had the appearance of a parasitic fibroid, but further investigation showed it to be situated between the layers of the mesentery of the lower part of the ilium. A small incision into it showed its contents to be of a *milky* appearance, which on further examination was found to have all the physical and chemical characters of chyle. The peritoneum was then incised over the centre of the tumour, and the cyst, the size of a child's head, enucleated. The divided edges of the peritoneum were then brought together by a series of Lembert sutures, and a glass drainage-tube put down to the seat of the wound. The patient made a satisfactory recovery. Microscopic examination of the cyst wall showed it to be composed of connective tissue with some sparse muscular fibres, but there was no evidence of any epithelial or endothelial lining.

In diagnosing mesenteric from pelvic tumours, it has been suggested that the patient be put into an exaggerated Trendelenburg position, in order to allow the falling away of the cyst from the pelvis, and to permit of an inferior zone of resonance being made out.

(Prof. Kynoch also asked permission to show an interesting specimen he had brought with him, and gave the notes of the case.)

HUGE RETRO-PERITONEAL FIBROLIPOMA.—The operation was exceedingly difficult, and when he was half through he wished he had not opened the abdomen. The operation was performed four weeks previously, and, much to his surprise, the patient had made a satisfactory recovery.

Dr Fordyce asked if the tumour was adherent to the liver?

Prof. Kynoch said to the left side of the diaphragm and right under the liver, he thought, but it was very difficult to say.

Dr Berry Hart said he must have got into the mediastinum.

Prof. Kynoch said he thought so.

Dr Fordyce asked if there was much bleeding.

Prof. Kynoch said almost none at all.

Dr Berry Hart said there was a big retro-peritoneal ovarian something like this figured in one of the Journals the other day.

Prof. Kynoch said, in course of removing the tumour he tore portions of the abdominal viscera.

The President asked if any of the Fellows had any remarks to make on this interesting subject.

Prof. Kynoch said there was nothing special about the history, the patient, æt. 46, had had two children. She had noticed the tumour three years previously.

Dr Haig Ferguson said Professor Kynoch was to be

congratulated on this operation. He had seen two such cases, one of which the President might remember—a case of Sir Halliday Croom's, and in that case, too, the specimen came to an untimely end because it was burned in the fire in Chambers Street. Dr Halliday Croom opened the abdomen—now about thirty years ago—thinking it was an ovarian tumour, and found it quite irremovable. The abdomen was closed and the patient lived for a year afterwards. She became greatly emaciated, as these cases do, and ultimately died. At the time of death the tumour weighed considerably more than the body of the patient. The specimen was kept in a huge tub in Minto House, and took two men to lift it.

Dr Berry Hart said he was present at that case, and that Sir Halliday Croom opened the abdomen and tapped the tumour, although he could not remove it.

Dr Ballantyne said he had never met with any chylous cysts in the abdomen. Within the last three years he had had two retro-peritoneal tumours, and these tumours gave the maximum of anxiety. The first was of cystic nature; the patient did well and continued well. He did not know how the marsupialised part had behaved as regards the intestine; it had caused no trouble until a fortnight ago, when he was asked to see the patient again, and found a swelling in the pelvis. He was afraid he would have to operate again. The second case was one of retro-peritoneal sarcoma, and instead of being content with marsupialising it, he worked away till he got at the tumour and drew off the fluid; but after getting down to it the patient died from shock. These were the only two retro-peritoneal cases he had had; these cases were always anxious, one never knew what tissues one was dealing with.

The President thanked Professor Kynoch on behalf of the Society as well as on his own behalf for the extremely interesting accounts of the two cases he had brought before them. There could be no doubt that these retro-peritoneal tumours

and mesenteric tumours were extremely rare; on the other hand, one frequently met with retro-peritoneal tumours arising from the ovary. He had seen three cases of mesenteric cysts. In two of his cases the cyst was removable, but in the third it was not, on account of the tremendous adhesions. Further, in the second case he had difficulty, because as the result of the removal of the tumour and the tearing of about a foot or 18 inches of the intestine, the patient on the tenth day suffered from absolute obstruction. On reopening the abdomen it was found that part of the intestine was, practically speaking, becoming gangrenous. It was necessary to resect more intestine to make things right; eventually the case turned out satisfactorily. That showed the many dangers which existed. The hæmorrhage was not so profuse as sometimes it was in these cases, and he was astonished that Professor Kynoch did not get hæmorrhage. Ovarian tumours splitting up the mesentery gave rise to a great deal of trouble.

Professor Kynoch thanked the President and the other speakers for their remarks.

V. ON CÆSAREAN SECTION AND REPEATED CÆSAREAN SECTION FOR CONTRACTED PELVIS, WITH NOTES OF A CASE SUCCESSFULLY PERFORMED FOR THE THIRD TIME ON THE SAME WOMAN.

By John M'Gibbon, M.B., M.R.C.P.E., Extern Assistant Physician, Royal Maternity Hospital; Assistant Gynæcologist, Hospital for Women, Edinburgh.

Cæsarean section has become in recent years an operation so extensively practised by the obstetric surgeon, and one with which he is so familiar, that I had at first some hesitation in bringing the subject before the Society to-night, recognising as

I do the frequency with which it is carried out, owing to the increased scope of its indications, and the success which attends at the present day its performance.

My desire to-night, however, is to ask your attention, mainly, though not exclusively, to the consideration of repeated Cæsarean section, as I am disposed to think there are some points regarding the repeated operation which merit discussion and criticism, and only within the past year the subject has engaged the attention of several of the important Obstetrical Societies of Europe and America. The history of Cæsarean section although full of interest, is so well known that to enter into it in detail would be an abuse of valuable time, but one might be permitted to recall a few facts relating to its earlier career.

Routh, in a most valuable and elaborate paper on Cæsarean section, read at an International Congress of Obstetrics and Gynæcology held at St Petersburg last September, states that the teaching of Mauriceau of Paris, whose work on the *Diseases of Women with Child* was translated into English in 1752, must have greatly influenced British obstetricians. There was certainly no mistaking Mauriceau's view, expressed as it was in the following terms:—It is a "damnable policy martyring and killing the mother to save the child," and advised that a living child be destroyed by embryotomy "rather than resolve upon that cruelty and barbarousness of the Cæsarean section, in which it is absolutely impossible that a woman should ever escape."

Mauriceau's doctrine apparently had the effect of holding British operators of the day in check, for during the next seventy years until about 1821 only a few were attempted, and all with one exception ended fatally.

The conservative operation from 1821 still struggled on until 1876, with a mortality of not less than 90 per cent. In 1876, however, Porro introduced his method of removing the

uterus after delivery, leaving a stump which he treated extra-peritoneally, by fixing it to the wound.

It was not until 1888 that Champneys of London and Murdoch Cameron of Glasgow did the first successful conservative operations, and from this moment onwards the Porro operation seemed to lose favour, while the conservative Cæsarean once more took its stand, now however under much more favourable conditions, as the rapidly diminishing mortality shows.

Thus in Glasgow in 1891 the mortality had fallen to 38 per cent., in 1902 to 20 per cent., and in 1904 to 12 per cent. To-day the mortality of conservative Cæsarean section, when performed for contracted pelvis, when all but septic cases are included, is about 6 per cent.; while in the favourable cases, where Cæsarean section is performed before labour, or before the membranes have ruptured, the mortality is about 2·9 per cent.

My own experience of conservative Cæsarean section has not exceeded six cases, all of which were for contracted pelvis, and successful to mother and child. Three of these were performed on the same woman, the only case on which I have had the opportunity of repeating Cæsarean section.

I shall give the history only of the repeated case:—

Mrs H. had her first confinement on the night of 12th February 1906. She had been in labour all day, and as little progress was being made, her doctor sent for me. I found the head in the L.O.A. position, not engaged, and powerful uterine contractions going on, dilatation not complete, and membranes ruptured. Under chloroform one recognised marked disproportion between the head and the pelvis, and I doubted the result of forceps, but attempted delivery with the patient in the Walcher position. A fair trial failing to bring the head through the brim, I performed craniotomy. A large well-developed child was delivered, the extraction even after

thorough comminution, and finally a double cleidotomy, being extremely difficult. The recovery was slow, but not complicated. Subsequent examination of the pelvis gave the following approximate measurements:—

Intercristal	$9\frac{1}{4}$ inches.
Interspinous	$8\frac{1}{2}$ „
External conjugate	$6\frac{1}{2}$ „
True conjugate (as estimated)	$3\frac{1}{4}$ „

It is only right to say that this patient had been carefully handled by her doctor before I saw her, the child alive, and I question if I was justified in performing craniotomy.

Second Pregnancy and Cæsarean Section I.—The patient became again pregnant in February 1907, and labour expected about middle of November. By careful examination under chloroform about the thirty-third or thirty-fourth week, one found the head could not be made to enter the brim, and I therefore determined on Cæsarean section as nearly as possible one week before full term. The patient, however, passed into labour two days before I had arranged for the section. The first stage had come on very rapidly, and as it was some hours before it was possible for me to see her, I found her on my arrival in active labour with nearly full dilatation, and preparations for the operation were hastened. This was performed then on the night of 6th November 1907. Uterus opened without eventration by median-longitudinal incision; placenta not encountered; bleeding slight; contraction active; puerperium normal; in bed four weeks. The child was quite healthy and vigorous at birth, but took a convulsion when three weeks old and died suddenly.

Third Pregnancy and Cæsarean Section II.—The third pregnancy was expected to reach term about 28th December 1908, and I hoped on this occasion to perform the section a week before full term. The section was performed on 22nd December 1908. A careful scrutiny of the uterine wall revealed absolutely no trace of the previous scar, and there were no uteroparietal nor uterovisceral adhesions.

Same procedure as in Section I.

Placenta not involved in the incision, and although no uterine contractions were going on as in Section I., the bleeding was no greater. Membranes a little troublesome to detach. The uterus was closed in the usual way, and I made no attempt to produce a uteroparietal adhesion by suturing it to the abdominal wall. Recovery uninterrupted.

Fourth Pregnancy and Cæsarean Section III.—The patient became pregnant about ten months after her second section, for the fourth time, and I performed the third Cæsarean probably about a week before labour, on the morning of 10th July 1910. On this occasion, judging from the experience of others, I expected to have to cope with extreme adhesions. To my surprise there were none. All that could be seen of the scar was a fine linear depression less than 2 inches in length, so that it was partially obliterated. The uterine incision I made along this linear depression.

I took special note as to the presence of any thinning of the muscle wall in the site of the old scar, but there was no evidence of any. Dr Haig Ferguson, who most kindly came to this section and who gave me his assistance, also carefully noted the entire absence of any thinning of the uterine wall in the scar region, as also the reduction of the scar to a fine and only partially defined depression.

The placenta was on this occasion anterior, but by making a small incision at first, just sufficient to introduce a couple of fingers, one was able to partially separate the placenta from the uterine wall and then rapidly enlarge the incision to the required length. Bleeding was greater on this occasion, but the uterus on being eventrated after delivery, responded fairly soon to hot saline swabs. After closing the uterus, sterilisation was carried out by ligature of the tubes in two places, and excision of the intervening portion. Recovery was if anything more rapid than on former occasions, the patient rising on the twelfth day.

And now one might here make a passing reference to the operation of induction as compared with an elective Cæsarean.

Take the case, for example, which I have referred to. The indication for Cæsarean section was of course only relative. It was a generally contracted pelvis, with a true conjugate of $3\frac{1}{4}$ inches. Might not the induction operation have been carried out and a living child capable of survival delivered by this method instead of by Cæsarean section? We know that the induction of premature labour, of British origin, has always been a favourite one in this country, and when performed about the thirty-fourth or thirty-fifth week is followed by a comparatively small fœtal mortality, especially if the delivery is effected spontaneously, for the moulding which the fœtal head can at this period undergo is fully appreciated. There are some obstetricians, *e.g.*, Whitridge Williams and Hastings Tweedy, who have entirely abandoned the operation of induction. One must remember that the maternal mortality is practically *nil*, while statistics regarding the fœtal mortality vary. Thus Herbert Spencer states that in 33 inductions for pelvic contraction, he had a 12 per cent. fœtal mortality, and Eden in 101 inductions had over 13 per cent.

Personally I have had a considerable experience of the induction operation, having performed it in successive pregnancies in a series of cases, with no fœtal mortality when these ended as spontaneous deliveries.

On the other hand, where, after the labour has been induced, and the head has either become arrested at the brim or been well engaged for some hours in the pelvis with no progress, and forceps have then been applied, I have had a high fœtal mortality. There must essentially be a time limit for the application of forceps in every case, whether it be a full-term labour or a premature one, in the interest of both mother and child.

I do not know precisely what percentage of cases of induced labour end spontaneously, but there are undoubtedly cases which require forceps, and the fœtal mortality in such

cases in my hands has been nearly 50 per cent. As regards the application of forceps, there must, as I have stated, in induced labours as in others be a time limit, but it is impossible to measure it in stated hours: it can only be properly measured in one way, and that is by good judgment on the part of the operator, which can only be acquired after years of experience. No matter how easy it may be possible to accurately measure a pelvis—and it is by no means always so easy—the relation of the size of the child's head to the size of the pelvis is of first importance; in other words, as Dr Barbour pointed out, the fœtal head is the best pelvimeter. If, then, at the thirty-fourth week the head cannot be pushed through the brim under an anæsthetic, the chances of a spontaneous delivery after an induction with survival of the child becomes a very uncertain matter, and this is where Cæsarean section becomes the operation of choice.

The Different Methods of Operating.

The usual method of conservative Cæsarean section introduced by Sänger a quarter of a century ago, and now so extensively and successfully practised, has obtained an important place in obstetrical operations.

Various operators proposed and practised different incisions of the uterine wall. Thus Cohnheim proposed an incision in the posterior wall; Caruso a saggital fundal incision, strongly supported by Morisani. The incisions, however, which found most favour were a longitudinal in the anterior wall, or a transverse at the fundus as proposed by Fritsch in 1897, and the two incisions for years vied with each other in the hands of continental operators.

Fritsch maintained that his transverse fundal incision made extraction easier, and was attended by less bleeding. It never was, however, so much employed in this country as the median longitudinal in the anterior wall, and some experienced

operators who have employed it, as for example Munro Kerr, finally abandoned it, not being impressed with the advantages which Fritsch claimed for it.

Quite apart, however, from the variations in the incision site of the modern intra-peritoneal Cæsarean section, the desirability of being able to empty the uterus by some method which would be purely extra-peritoneal, began to engage the attention of obstetricians.

Nurnbergen points out that the idea of extra-peritoneal section was proposed so long ago as 1806 by Jorg, and actually performed by Ritgen in 1821. Subsequently Gaillard Thomas, of New York, in 1870 performed a similar operation—laparo-elytrotomy. The method did not, however, attract real attention until Frank of Cologne published his first cases in 1907. Since Frank described his first operative procedure, many modifications have been made upon it, and one does not wish in this paper to do more than indicate the salient points of the more important of these. During the past three or four years the extra-peritoneal Cæsarean operation has been extensively practised for contracted pelvis, placenta prævia, eclampsia, and just recently it has been advocated by Russell of Glasgow for transverse presentations in contracted pelvis.

The original aim, however, of Frank, who introduced it, was to be able to empty the uterus in any case which seemed likely to have become infected, without contaminating the peritoneal cavity by any discharges which might escape into it from the uterus during the process of delivery. Whether such original aim of Frank and of those who followed him has been realised, we shall refer to later.

Frank's Operation.—With raised pelvis, a transverse incision is made through the whole abdominal wall, a little above the symphysis. The recti are widely separated. The peritoneal cavity is opened temporarily, and the peritoneum is stripped from below upwards off the upper surface of the bladder and the

anterior wall of the uterus. The cervix is then incised transversely, and the child delivered. This was the first attempt at extra-peritoneal section. The next method was that of Sellheim.

Sellheim's Method.—Transverse incision through superficial parts. The deeper parts opened by a longitudinal incision. Having opened the parietal peritoneum, the visceral peritoneum where it passes from the bladder on to the anterior wall of the uterus is divided transversely. The upper flap is freed by blunt dissection, is stripped up, and is temporarily stitched to the parietal peritoneum at the upper end of the abdominal wound. If necessary the lower peritoneal flap is stripped off the bladder and turned down. The cervix being laid bare is now incised longitudinally. The bladder was filled with sterile fluid to define its contour.

In 1908 Latzko and Döderlein devised methods for gaining access to the cervix without opening into the peritoneal cavity at any stage, truly then extra-peritoneal all through. The following is the usual description of the Döderlein, or as it is sometimes referred to as the Latzko-Döderlein, method. The bladder is well injected to define its contour.

A transverse incision with separation of the recti and partial detachment of the right rectus near its base. The hand is then introduced to the right of the bladder (which being distended is easily defined), the loose connective tissue separated, by means of which the anterior surface of the lower uterine segment is exposed. There is no detaching of the peritoneum from the bladder. The head usually pouches the lower uterine segment forwards considerably, and at the top of this part which pouches forwards, a longitudinal incision is made and carried downwards, and delivery effected.

This seems at the present moment to be the method most generally employed on the Continent, and has recently been carried out with approval and success by Russell of Glasgow, by Hastings Tweedy, and other British operators.

An endeavour to successfully cope with cases which the obstetrician believes must be septic, has been lately carried out, apparently with success, by a manœuvre suggested by Sellheim. This is to establish a utero-abdominal fistula. Briefly, he opens the abdomen with a longitudinal median incision, stitches the skin to the parietal peritoneum, incises the uterine peritoneum longitudinally, and strips it off the line of the intended uterine incision, sewing it also to the parietal peritoneum. The uterine contents are extracted by a longitudinal incision, the uterus being left open, the edges being stitched to the edges of the abdominal wound, thus establishing a utero-abdominal fistula, which Sellheim maintains contracts rapidly as a rule, and only occasionally requires a subsequent plastic operation.

Having reviewed in brief the most recent methods of continental fashion in the performance of abdominal Cæsarean section, where are we? That is the practical point for the obstetric surgeon to face. Are we in cases of contracted pelvis to abandon the time-honoured classical section of the corpus uteri, and instead deliver from the lower uterine segment by some form of extra-peritoneal route? Frank and his train of followers stated that it was to meet the requirements of septic cases that the extra-peritoneal operation was devised, and maintained that its great advantage in all such cases was the prevention of contamination of the peritoneum by infected uterine contents. But has this advantage, enormous as it would have been, and meaning the saving of the patient's life, been actually realised? I do not think it has. Another advantage widely claimed for it was that there was less danger of hæmorrhage; but every operator experienced in the classical operation knows how seldom is the bleeding formidable.

The extra-peritoneal methods have been by no means free from trouble, and an inquiry into the experiences of different operators substantiates this. Frank himself admitted that in cases where the lower uterine segment had not been extended,

hæmorrhage had been very considerable, also that detachment of a large area of the peritoneum had caused gangrene.

Hæmorrhage is prone to occur from the vesical veins, and on several occasions has been so severe that the operator was compelled to abandon the extra-peritoneal operation, and perform instead the ordinary classical operation. Such has been the experience of Fraipont, Franz, and many others. Kustner also speaks of the difficulty he has encountered in the extraction of the child, and like some other operators uses a pair of small forceps for extraction. Lacerations of the bladder and severe tears of the peritoneum were not infrequent. These, however, appear to have been of less frequency in the Döderlein method.

The mortality of these extra-peritoneal operations has been variously stated. By some it is given as 5 to 7 per cent., but further experience tends to make it higher. The morbidity is about 30 per cent. The fatal cases are generally the result of peritonitis, in spite of the fact that the peritoneal cavity is supposed to be shut off from the operation area.

H. F. Lewis, however, collected 102 cases, and finds the maternal mortality of extra-peritoneal Cæsarean section to be 8·8 per cent.

He asks the question: "Is this cervical Cæsarean section really better adapted for infected cases than the other means at our command?" He concludes that it is not proved that it is safer than ordinary Cæsarean section for the type of case for which it was primarily intended. His reasons are:—

(1) The peritoneum is much more resistant to infection than are the cellular tissues.

(2) Proper packing of the general abdominal cavity ought to greatly diminish chance of infection.

(3) It is a more difficult operation and opens up large connective tissue spaces.

(4) Extraction is often difficult.

This opinion of Lewis, expressed in 1909, has been corroborated by other writers since, and the opinion seems to be gaining ground that both trans-peritoneal and extra-peritoneal section is a dangerous operation in septic cases, the very type of case for which it was at first so much vaunted.

It does not seem to offer a better chance in infected cases than would the classical operation with careful shutting off of the peritoneal cavity. Lewis remarks in his paper that as gynæcologists we open the abdominal cavity frequently in the presence of virulent infection, and that we are usually able to shut off the general peritoneal cavity from contamination by abscesses, or wounded bowels, which are surely as septic as the contents of a fœtid amniotic sac.

Sellheim's suggestion of a utero-abdominal fistula should be borne in mind, and worthy I think of trial in a really septic case. Routh is convinced that in a recent case he had, a utero-abdominal fistula was the important factor in saving the patient's life. It seems to me the difficulty in the past has been to be sure of the presence or absence of sepsis, as in some cases the danger signal is not in evidence at the moment when we are called in to decide the course to be followed. There are many cases I am sure just on the borderland, and these have been the difficult ones to deal with.

Now, however, there is the hope that a bacteriological examination of intra-uterine swabbings may shortly prove of invaluable help to us in identifying, even within an hour, the presence of such organisms as would deter most of us from performing a Cæsarean section of any kind, and thus place such an important decision on a precisely scientific basis.

Given, then, a case of contracted pelvis where infection is proved, and we refuse abdominal section, craniotomy is the only alternative. Within recent years there has been an outcry against this procedure, and justly so. There are obstetricians who will not perform it under any circumstances. Pinard is

one, and his death-rate in such cases is 20 per cent. Munro Kerr says, with reference to his refusal to perform Cæsarean section in cases which have been interfered with before coming under his own notice: "I am compelled not infrequently to perforate a living child. It is always with extreme regret, but I am perfectly convinced I save more mothers, and probably indirectly more children." With that I quite agree. We cannot yet make a clean sweep of craniotomy, for the object in performing it in infected cases is that we can empty the uterus of its contents under antiseptic methods, and avoid the development of acute peritonitis from the uterus in a Cæsarean section.

We must bear in mind, however, that in the vast majority of such cases prolonged attempts to deliver by the forceps have been made, and much bruising and laceration of the parts is the result. Add to that some degree of abrasion while performing the operation, and it is no small wonder that craniotomy performed in already infected cases, should have much morbidity and a death-rate of about 10 per cent.

Routh points out, however, that the classical Cæsarean section performed under such circumstances even within the last ten years was not less than 34 per cent. The mortality of craniotomy then, performed on infected cases, is very distinctly (nearly 20 per cent.) less than than of Cæsarean section performed under the same circumstances. In the tables presented by Routh it is impressive to note that of 216 cases where attempts at delivery had been made, and where conservative section was done, 40 died, whereas in 14 cases where the uterus was removed by hysterectomy, leaving the stump for intra-peritoneal treatment, not one died.

At the present day a pan-hysterectomy would find favour with some, with the idea that the cervix being septic might be a source of infection; but the risk of infection of the peritoneum from the septic vagina after removal of the uterus is difficult to prevent.

Referring to the employment of the various operative procedures in suspected cases of infection, Pfannenstiel doubts whether such should be adopted merely to save a child already in danger from infected liquor amnii, and adds, "I believe that our modern attempts to save the child's life shoot beyond the mark."

The Production of Adhesions, Accidental or Artificial, and their Effect.

The production of adhesions as the result of a primary Cæsarean section, has for long been a source of anxiety to the operator, and has been regarded by some of such serious import as to justify the performance of sterilisation at every Cæsarean section. Such adhesions have time and again led to disastrous results, and from various causes. They may be so dense and firm that their separation causes severe bleeding, or the intestines may be wounded or actually perforated during the process, and such complications of course necessitate not only the greatest surgical skill, but they increase—in some cases very considerably—the duration of the operation, and thus transform one of the simplest abdominal operations into one of extreme difficulty and danger.

Not only has one to consider the development of omental and intestinal adhesions, and their dangers, but also those which form naturally between the anterior wall of the uterus and the abdominal wall—the utero-parietal. Experience has taught us that by mere chance, adhesions may form between the uterus and the abdominal wall, so firm and so complete, that the operator may at a repeated section, open straight into the uterus without at any point entering the peritoneal cavity. Both old and modern operators have referred to this utero-parietal adhesion, and many reports of such are to be found in the literature. One of the most remarkable of these was placed on record so long ago as 1836 by Michaelis of Kiel, who

performed a Cæsarean section for the fourth time on the same patient without entering the peritoneal cavity. The same has been the experience of Sinclair, Wallace, Fruinsholz and Michel, Green, and many others. Various factors no doubt are at work in the production of such natural adhesions. Paul Bar said in 1900, that he considered the formation of such adhesions indicated defective asepsis, and believed that with more perfect technique, adhesions would disappear.

It is not possible to entertain such a proposition, for we know that mechanical injury, chemical irritation, the mere presence of sterile ligatures, produce within the peritoneal cavity an effusion of lymph, to which any peritoneal surface may become adherent, and in this connection many cases which have undergone a primary section, and have made a perfectly aseptic recovery, have shown evidence of pronounced adhesions at the second or third operation.

Wallace of Liverpool, who published an interesting monograph on repeated Cæsarean section, representing a most exhaustive investigation and calculated to throw much light on the whole subject, deals very specially with the question of adhesions. In 60 modern cases he found that in the report of 17 no mention was made of the presence or absence of adhesions, while in 43 performed by different operators he found a note of adhesions had been made. He tabulates them as follows:—

A.	Cases with incomplete utero-parietal adhesion .	14
B.	„ „ complete „ „ „ .	8
C.	„ „ very slight „ „ „ .	4
D.	„ „ utero-visceral adhesions . .	10
E.	„ „ no adhesions	7
		43

Of those 60 repeated Cæsareans, there were 4 deaths, and all 4 were cases of utero-visceral adhesions.

Recognising that in those cases where a complete utero-parietal adhesion had incidentally formed, the operation became simple, easy, and safe, Sir William Sinclair attempted to ensure by special procedure the formation of a complete utero-parietal adhesion in every case. He endeavoured to produce this result by rubbing iodoform over the anterior uterine wall combined with padding and firm bandaging of the abdominal wall.

Other operators had endeavoured to produce utero-parietal adhesion, but not with a view to future pregnancy, their object being solely to prevent leakage from the uterus into the peritoneal cavity.

It was A. J. Wallace of Liverpool who suggested that the most certain way to produce such a utero-parietal adhesion was to fix the uterus by sutures to the abdominal wall. Sinclair and Wallace in different papers both describe the details of the technique carried out. Briefly stated, it is an extensive ventrofixation produced after closure of the uterus, by a series of sutures introduced through the parietal peritoneum on one side, taking then a grip of the uterine wall about half an inch outside the uterine wound, and then on through the parietal peritoneum on the opposite side of the wound. Sinclair also introduces one or two silk sutures through the fascia and peritoneum, which take a good grip of the uterus and pass out through the peritoneum and fascia on the opposite side. He points out that the production of such a procedure—

(1*st*) Ensures a wide area of adhesion.

(2*nd*) That it supports the uterus and prevents its displacement during involution.

(3*rd*) That it excludes omentum and intestine from the field of future operation.

Now if such a utero-parietal adhesion could be invariably relied upon to preclude from the field of future operation omentum and intestine, and if at the same time it were to be

followed by no serious consequences during a future pregnancy, it appears to me we should have here—so far as repeated section is concerned—one of the simplest and safest of all the extra-peritoneal operations to perform.

Attempts, however, to produce such an adhesion have met with adverse criticism by many writers, who all refer to the same proposition, viz.—that if the anterior uterine wall be fixed, it has little chance of expanding during the growth of pregnancy, and that in consequence the uterine enlargement must necessarily take place in the posterior wall, meaning thereby its thinning and its tendency to rupture, the firmer the adhesion the greater the tendency. Here, however, we may remark that the operator of to-day when dealing with a patient still in the child-bearing period, takes care when performing ventrofixation, to do so well down the anterior wall, leaving the fundus entirely free to expand if pregnancy supervenes.

In Routh's collection of 1282 Cæsarean sections, I find only six reported to have had a ventrofixation performed, and in subsequent pregnancy, no actual rupture occurred, although in one of this series reported by Targett, and one by Jardine, it was evidently threatened. Are we, then, to abandon the method suggested by Sinclair and Wallace of providing for an extra-peritoneal operation in a future section, as predisposing to rupture and possible strangulation?

Some Points in the Technique of the Ordinary Classical Operation.

The careful closure of the uterine wound is an absolute essential if one has regard to the influence of a future pregnancy. The older operators never closed the uterus at all, and still many of their patients recovered—often, however, with utero-abdominal fistulæ, which, may I suggest, was in those days probably their safety-valve, for it will be remembered that one of Sellheim's latest suggestions for septic cases was to

create a utero-abdominal fistula. Such, however, must inevitably leave a weak cicatrix prone to give way under the strain of a future pregnancy. Wallace says: "Not mere healing, but total obliteration of all traces of the wound should be aimed at," and he advises that three layers of sutures be used. The first he says includes decidua and part of the muscular wall. This means that part of the suture lies exposed in the uterine cavity, and to this many operators object. The second layer, he says, should include muscle only; and the third, muscle and peritoneum together. Whatever be the operator's method, it is desirable to cover the deep sutures, by overlapping the peritoneal edges.

As regards the suturing, may I here say that I have found for the passing of the deep sutures nothing to equal Doyen's curved uterine needle. It is ideal for this purpose. It is doubtful if it matters very much whether silk, catgut, or linen thread be used. Personally I have stuck to catgut in my own cases. It is Sinclair, however, who strongly advocates silk for the uterine wound, and says: "My belief is that if silk were always employed that we should seldom or never hear of rupture."

As regards the suturing, it seems to me that in including the decidua in the first layer of sutures, there is both an advantage and a disadvantage. If one goes right through the entire substance of the uterine wall, one is more likely to obtain complete union of the entire muscular layer, a most important aim; while, on the other hand, if the contents of the uterus be not aseptic, one here establishes a continuity between decidua and peritoneum which might quite well be responsible for peritoneal infection. If one is dealing with a presumably aseptic uterus, I am quite convinced one should go right through the whole uterine wall, for by so doing, one prevents the inner and deeper edge from retracting, which it is very apt to do. On the other hand, M'Pherson of New York believes it

is essential not to include the mucous membrane with the sutures at all, not merely for fear of infection, but also because he believes that the mucosa may become included between the edges of the muscle, and such foci of mucous membrane may in successive pregnancies be transformed by the ordinary method into decidual tissue, thereby leading to separation of the walls and subsequent weakness of the scar, thus allowing the uterus to rupture. In all probability, he says, many ruptures are due to this cause. Now I admit one cannot deny the possibility of such an occurrence, but so far, few cases have been produced to prove it, whereas we have a good many cases recorded in which it was clearly shown that complete union of the muscular layer had never taken place, and I feel disposed to regard the latter as a much more likely predisposing cause of rupture than the former.

The Relation of Repeated Section to Rupture.

The danger of the uterine cicatrix giving way during a future pregnancy or labour is one of the leading arguments of those who are opposed to the performance of repeated Cæsarean section. Both after the transverse fundal incision, as also the longitudinal median, has the resulting cicatrix given way at a future pregnancy or labour. Thus rupture occurred after the fundal incision in cases recorded by Munro Kerr, Ekstein, Schink, and more recently by Mabbot, Dahlman, Nacke, and Meyer of Copenhagen.

Of a series of 26 cases collected by Dahlman, 13 are said to have followed a transverse scar. On the other hand, Simrock of Bonn records 54 cases performed by Fritsch's transverse incision, of which 3 had the operation performed for the second time, and in these he found a strong cicatrix, with no evidence of thinning of the wall or tendency to rupture. An examination into the literature of scar rupture after Cæsarean section shows that a large number have been placed on record, but it

has been disappointing to find the reports lacking in important details. A considerable number of cases are on record following the longitudinal scar on the anterior wall, and occurring through it. Such are reported by Kobauk, Woyer, Guillaume, Targett, Everke, Galabin, Werth, Schneider, Lobenstine, Brodhead, Brothers, Prüsmann-Henkel, Convelaire.

Richter, Scheffzek, and Jeannin give full reports of recent cases, but in addition to the foregoing there have been other cases, the reports of which are very incomplete.

As regards the exact frequency of the occurrence of rupture in relation to repeated section, reliable statistics have been difficult to obtain. Olshausen, for instance, had only met with one case of rupture in 120 sections.

Convelaire thinks rupture occurs probably in about 2 per cent. of cases, whilst the mortality of repeated section seems to be 5 to 6 per cent. In 150 cases collected by Pollok, it was only 5 per cent.

The rupture may occur during pregnancy or at the onset of labour, but far more frequently after labour has been in progress for some time.

Of 20 cases collected by Brodhead, it is interesting to note that only in 5 is it definitely stated that rupture occurred before labour had set in. Further, in at least 3 of this series, there was evidence at the second Cæsarean section to demonstrate that the uterine wound of the previous operation had failed to unite through the entire thickness of its wall. It is quite possible that such a condition of affairs may have been present in other cases of this series, but whether or not, 15 per cent. of uterine wounds found imperfectly healed only goes to prove what I should like to lay stress on, viz., the absolute necessity of spending a little extra time and care in endeavouring to secure perfect coaptation of the uterine surfaces.

I find that Sinclair, Munro Kerr, and Wallace all refer to

the importance of this. I feel convinced that if thorough apposition be secured, with a minimum of cicatricial tissue, the cases of rupture will be few to record.

Mason and Williams performed a series of experiments on pregnant animals, the object of which was to ascertain certain points relative to uterine rupture in pregnancy. They performed Cæsarean section on these, and killed them after periods of not less than full-term gestation. They made comparative tests of the muscle and scar respectively, by applying weights to a part of the uterine wall containing both. In every case the muscle gave way first, and in one instance only did the rupture extend secondarily into and along the scar.

Two animals became pregnant for the second time. Cæsarean section was repeated, and near term the test was made again, the result being the same as in those who were not again pregnant, thus ruling out any change in the strength of the scar during pregnancy.

Their conclusion is that a carefully sutured and well-united scar will stand any strain which can be endured by the uterine muscle, and they maintain that rupture of a scar is always secondary to unusual weakness of the scar, and dependent upon imperfect consolidation. The most frequent cause of imperfect consolidation is placing the deep sutures too far apart, or not including the entire thickness of the uterine wall, or from infection of the scar.

I do not wish, however, to infer that even after the most careful surgical technique, rupture will never occur, but I believe it should be rare. Richter, for instance, recently reported to the Dresden Gynæcological Society a case where he said a most thorough repair of the uterine wall had been carried out, and yet rupture occurred at the fourth month of the subsequent pregnancy. This, I should think, must almost be unique at the fourth month. Jeannin and Wilhelm also

showed at the Paris Obstetrical Society, a uterus which had ruptured at the scar of the previous section. The patient was admitted to hospital in labour, but they believe rupture occurred after admission while she was being prepared for craniotomy. They found no evidence whatever of imperfect union of the uterine wound, nor of thinning of the wall, nor was there even any distension of the uterus. Allowing, then, that such cases do occur even after the most careful technique, there must be some other etiological factor at work.

It was Kaltenbach who first suggested that the cause of rupture depended on the presence of infection of the uterine wound. Certainly in some cases of rupture there was evidence of suppuration having occurred in the wound with involvement of the uterine cicatrix.

Werth maintains that it must sometimes be due to the rhythmic contractions of the uterus, preventing an exact union of the freshly sutured wound surfaces, for in a case in which the most exact suturing was performed, and in which rupture occurred, it was found that only very thin strips of muscle formed the union, appearing as a very thin layer under the peritoneum.

Karl Hartmann refers to a case of rupture where the tear had caused the edges to become everted, demonstrating, he says, the pull of the muscular fibres.

Sterilisation.

In the absence of absolute indications—as, for example, fibroids, malignant disease, or sepsis—under what circumstances are we justified in sterilising a woman while performing Cæsarean section for pelvic contraction? Few questions, especially from the ethical standpoint, have been the subject of greater controversy than that of sterilisation. Without wishing to be dogmatic, I hold that in the absence of pelvic or other disease, or any indication save the contraction itself, no

woman ought to be sterilised, and no operator should encourage such a procedure at the first section. Green, in a paper read before the American Gynæcological Society, says: "I am more strongly convinced than ever that it is not justifiable to sterilise a woman even if she and her husband request it." To that statement most of us would, I presume, take exception. I feel strongly tempted, however, to agree with Green in so far as such a statement is made applicable to the first section. The child delivered at the first section may die, as happened three weeks afterwards in my repeated case. Now if I had sterilised that patient at her first section, to which she and her husband would have had no objection, the circumstances—no child, and being sterilised—would, as I afterwards learned, have caused bitter regret.

I am quite certain that women who desire sterilisation at their first section, do so for the most part under an unnatural dread of its performance being repeated. They regard it almost as a tempting of Providence. Now in view of the evidence which can be led before us to-day relative to repeated sections, which evidence goes to prove that after all, the repeated operation is attended by if anything a smaller mortality than the primary, I submit it is the duty of every operator to discourage sterilisation at the first section. At the same time it becomes his imperative duty to point out to every woman and to her husband, the absolute necessity of reporting her condition to her doctor during the earlier months of pregnancy. If a woman does not do so, then she deliberately neglects the most important step towards her self-preservation, for which no surgeon can be in the slightest degree responsible. The importance of this cannot be laid down too strongly, for there is abundant evidence to prove that rupture—the dreaded danger—takes place in the vast majority of cases after the onset of labour. Here lies the importance of giving the surgeon the chance of performing an elective Cæsarean.

Of the 150 cases reported by Polak of New York:—

116 had Cæsarean section performed twice,
32 „ „ „ „ thrice,
1 „ „ „ „ four times,
2 „ „ „ „ five times.

and yet the mortality was only 5 per cent.

M'Pherson gives satisfactory information regarding thirty-nine repeated Cæsareans, and representing 120 operations:—

30 had Cæsarean section performed twice.
7 „ „ „ „ thrice.
1 „ „ „ „ four times.
1 „ „ „ „ five times.

It is interesting to note that in this series, 29 cases showed few or no adhesions; in 7 there were many; and in 1 only was the uterus adherent to the abdominal wall. In 1 only was rupture met with.

In this series of 120 operations, there were only three deaths.

Such records, I think, are sufficient to satisfy our minds that the repeated operation offers very little increased danger, if any, and a smaller mortality when compared with the primary, and I therefore submit that except under very special circumstances, it is not the duty of the surgeon to advocate sterilisation to those patients who come under his care for repeated section. Dr Munro Kerr, who has had an exceptionally extensive experience, having performed up to date close on 90 Cæsareans, says in his *Operative Midwifery*: "It has been my practice up to the present time (1908), to sterilise *all* patients after a second Cæsarean section, for I feel that a woman who has twice subjected herself to Cæsarean section, has done sufficient for her family and the State." Notwithstanding the fact that such an experienced and successful operator should come to this conclusion, I cannot agree with

him. I think we are fully justified in performing the operation a third, fourth, or even fifth time, if the patient herself, having had previous experience of the operation and having the possible dangers laid before her in a fair way, should desire delivery by section. My own patient and her husband urged me at the third section to perform sterilisation, which I did, reluctantly however. I entirely disagree with Green, Herbert Spencer, and others, who maintain that the patient's wishes should not be consulted. I am of opinion that we are fully justified in advising a woman to a third or further Cæsarean section, but under no circumstances justified in refusing to perform sterilisation if she and her husband demand it. This may be performed in a variety of ways, as by ligature of the tube in two places and excision of the intervening portion. This has the possible advantage that if the patient changed her mind and desired another child, the divided ends might be reunited and the patency of the tube restored. This ingenious suggestion was made, I think, by Dr Munro Kerr, but whether he or any other operator has ever carried it out, I do not know. Probably the surest way is to thoroughly excise the tube from the uterine wall.

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The President said they would be glad to hear any remarks on Dr M'Gibbon's practical paper.

Dr Berry Hart said Dr M'Gibbon had raised a very great many points, and his paper would have been improved if it had been more condensed. He was to be congratulated on the fact that he was able to perform Cæsarean section on one woman three times successfully. The difficulty in Cæsarean section

was, of course, the infective case. In these cases one should get a really good bacteriological report. The infection could be carried through the cellular tissue as well as through the peritoneum. He did not agree with Dr M'Gibbon about going right through the uterine wall. He (Dr Berry Hart) had first successfully done Cæsarean section in Scotland. The first really successful operator was a cow in a grass park! One could not make deductions from statistics.

Professor Kynoch said he had listened with great pleasure indeed to Dr M'Gibbon's paper, but felt inclined to agree with Dr Berry Hart that he had perhaps included too many of the more important aspects of the operation, each one of which might have formed a discussion by itself. With regard to the technique of the operation, especially for extra-peritoneal Cæsarean section for infective cases, he had nothing to add, not having had any experience of them. For contracted pelvis he had invariably done craniotomy; it might be, however, that Cæsarean section was quite good; at the same time he thought craniotomy a good operation. The point that interested him most was the question of sterilising the patient after doing the operation once. It was only now when the operation of Cæsarean section was performed with such excellent results that the safety of the operation was being pointed out and the desirability of exposing the patient to repeated Cæsarean section advocated. He had not much experience, for he had not often met with cases of badly contracted pelvis. He had only had 8 cases, and in every one he had sterilised the patients. Now, however, as one's experience of the Cæsarean operation increased, one felt inclined perhaps to give the patient the chance of exposing herself to Cæsarean section a second time. He did not, however, think the patient should be exposed to the operation more than twice. Dr M'Gibbon referred to a patient in whom it had been performed four times successfully; but he did not refer to the well-known German case where it

had been done four times, and the fifth time the patient died from septic peritonitis. He thought after a second Cæsarean section the patient ought to be consulted. In cases of contracted pelvis the intelligence of the patient was usually pretty low—rickets as a rule was indicated—but he did not think one was likely to meet with a woman who would not understand the meaning of this operation after having had it done twice. In illustration he mentioned a case which had occurred two months previously. The patient was the wife of a working man, and the question was whether premature labour or Cæsarean section was to be done. The patient was a small, rickety woman with a conjugate of $3\frac{1}{4}$ inches—a very good case for premature labour. He pointed out to the patient and her husband that the chances for the child after premature labour were not good, whereas if Cæsarean section were performed the chances for the child would be quite good, and that it would not be necessary for the mother to be exposed to the operation again. The husband preferred Cæsarean section if the woman would not be subjected to it again. He thought in a case like that the matter ought to be left to the patient. This case would be operated on in the course of a fortnight, and he was going to do what the husband desired and sterilise the patient. If the patient had said, "Well, Doctor, I will leave it to you," he would have been inclined to leave it, and then if pregnancy occurred again to have performed Cæsarean section; but since the husband had decided against premature labour and for Cæsarean section, provided it should not be repeated, he felt justified in sterilising the patient. The chief danger seemed to be the risk of adhesions. If he did Cæsarean section with the intention of repeating it, he would be inclined to go in for some fixation method. The entire absence of adhesions in Dr M'Gibbon's case was interesting. He understood the danger in these cases was adhesion to the omentum and surrounding parts, rendering the operation more serious. Then,

also, the question of rupture, through a cicatrix of a former Cæsarean section, had to be kept in view. Dr M'Gibbon pointed out that if the wound be properly sutured one should not hesitate to allow the patient to go on a second time. He (Prof. Kynoch) did not agree with this, he did not hold with the view that on no account should a patient be sterilised. Personally he would not submit the patient to Cæsarean section more than twice. There were a great many other points in the paper—technique, suturing, and suture material—which would take a long time to discuss. He had listened with great interest to the paper, and thought Dr M'Gibbon had practically covered the whole subject.

Dr Haig Ferguson said he had listened with much interest to the paper, and agreed with the other speakers that there were a great many points which might be discussed. With regard to the question of the dangers of premature labour as compared with elective Cæsarean section, he would choose Cæsarean section if premature labour had to be induced very early. He was interested to learn of the sweeping condemnation of premature labour by certain obstetricians. In infective cases, he had no experience of the extra-peritoneal operation. He had operated on an infective case and the patient had done quite well. There were two things that might be done in infective cases—two suggestions had recently been made, namely,—(1) washing out the uterus with some saline before opening the abdomen—a method suggested by Maxwell—and this had been done with comparatively good results; and (2) that the placenta should be removed *per vias naturales;* the fœtus alone removed by the abdomen; and great care taken to see that none of the liquor amnii got into the wound. Professor Kynoch had indicated that if he intended to repeat the operation, he would be inclined to produce utero-parietal adhesions by some fixation operation. Sinclair of Manchester, and Wallace of Liverpool had been the first to

suggest ventral fixation of the uterus, so that one could remove the contents without opening the peritoneum. Now adhesions in the abdominal cavity, he believed, were always a source of possible danger. To deliberately form adhesions was a very unjustifiable thing to do, and he regarded such a procedure as a remnant of antiquity.

Dr Ballantyne said he would confine himself to one or two points. He had not done the extra-peritoneal operation, but he had written an elaborate summary on the subject for the *Edinburgh Medical Journal* a year and a half ago. After finishing that summary he felt perfectly certain that this operation would not often be chosen, partly from the fact that there were so many different methods of operating, and partly because the details for carrying them out were so variable. With regard to the infective cases, he had a letter recently from Dr Routh, who repeated the warning: "Do take a swab before you do anything." There was always the difficulty, however, of getting the bacteriological report in time. A practice which had been adopted in America and suggested by Davis was to make a supra-umbilical skin incision in Cæsarean section, and Davis reported a number of cases, but it was a little difficult to understand how it was carried out. It certainly had advantages if it could be done, and he (Dr Ballantyne) would like to try this method if he had the opportunity. A curious complication occurred in one case he had in the Maternity Hospital. The skin was prepared by the iodine method and healed very well; a couple of days later, however, a stitch abscess occurred. A stitch abscess so late must be a very exceptional thing. The four or five Cæsarean sections he supposed belonged to the same category as the old Roman historian who gave the account of the twelve Cæsars!

Dr Fordyce said he wanted to say a few words on a question which had not been touched on, namely, infective cases. He

supposed by an "infective" case was meant one which had been infected with discharge. Just a few days previously he had a case in the Maternity Hospital where the patient was subjected to forceps. The head was well above the brim, the child was alive, and, following the rules laid down, craniotomy was done but very much against his will. Personally he would not have had the slightest hesitation in performing Cæsarean section on that case. He thought the peritoneum was more able to withstand septic infection than any other part. If these cases were already infective they would be just as likely to die with craniotomy as with Cæsarean section. But there was the further point that Cæsarean section was done presumably to get a living child, with the greatest safety to the mother. In many of these cases the child had been subjected to a severe forceps operation, and if a Cæsarean section was performed the child would very likely die soon after labour.

Dr Lamond Lackie said he had done Cæsarean section twice in two cases, and had been struck with the fact that there was absolutely no trace of a scar at all at the second operation; in neither case could one make out even a linear cicatrix. With regard to sterilisation, he did not in the slightest degree agree with Dr M'Gibbon. One Cæsarean section was quite sufficient. He thought the woman should be consulted, and that the husband had nothing to do with it. With regard to technique, he was surprised that Dr M'Gibbon should advocate the introduction of stitches through the entire uterine wall. Had Dr M'Gibbon taken the size and weight of the child? He would like to know if the child happened to be specially large, because a pelvis of $3\frac{1}{4}$ inches was not very small.

The President said he thought, as the hour was late, they might conclude with this subject. There were two points, firstly, infective cases; and, secondly, the question of sterilisation. As far as infective cases were concerned, he had never

done extra-peritoneal section. He would either follow Dr Fordyce's advice and do craniotomy, or be disposed to do the usual Cæsarean section operation. In the early days of Cæsarean section they did not go in for the operation from choice but of necessity, and in all these cases the pelvis was small; the chances of craniotomy were so bad that one was driven to Cæsarean section. On each occasion the Lawson-Tait method was carried out; it was a simple operation, and anybody could do it. Uninterrupted recovery resulted. Many of these cases must have been infective. Extra-peritoneal treatment of the stump was the method indicated in certain of these infected cases. As regards sterilising he entirely agreed with Dr M'Gibbon, and did not agree with Professor Kynoch. He had done the operation of Cæsarean section three times on one woman, and hoped to do it a fourth and a fifth time, and the patient hoped so too. Considering the morality was about 2 per cent.—a 50 to 1 sporting chance—he was perfectly certain that many women would risk Cæsarean section. Many would risk a 5 to 1 chance, far less a 50 to 1 chance, to have a living child. The facts should be put clearly before the woman, and she should be told that the mortality was probably about 3 per cent., and the decision should rest with her entirely. He did not think the doctor had reason to give any advice in the matter. The subject should be treated just as one would treat cases of painful ovaries. They must all have learned something, he thought, of the subject from Dr M'Gibbon's paper, which was both practical and extremely interesting.

Dr M'Gibbon in reply thanked the President and the various speakers for their remarks. With Dr Berry Hart he agreed that the cellular tissue was just as likely to be infected as the peritoneum. He was exceedingly sorry that Professor Kynoch had sterilised his eight patients. He was much interested in utero-peritoneal fixation. When Sinclair and Wallace introduced that method it was adversely criticised; of all the extra-

peritoneal methods it seemed the most simple, and the only objection was the risk of possible rupture. If by fixing the uterus to the abdominal wall one was exposing the patient to rupture in subsequent pregnancy, then one was adopting a very dangerous procedure indeed. If Dr Haig Ferguson was going to allow the pregnancy to continue till within three weeks of full term, it seemed rather late for the induction of premature labour. He also mentioned a very important point with regard to the removal of the placenta *per vias naturales* which he (Dr M'Gibbon) had not thought specially of; it was a point which ought to be considered. Dr Ballantyne referred to the high incision, called the small high-medium incision. He (Dr M'Gibbon) like Dr Ballantyne felt perplexed as to how one was to deliver the child and the placenta; such an incision did not seem likely to make the procedure easy. He agreed with Dr Fordyce, that if the case had not been handled the indication was certainly to do Cæsarean section; but if there was infection, craniotomy was the operation.

Dr Fordyce asked what was meant by infection?

Dr Berry Hart said it did not mean anything. One must have a pathogenic condition, streptococci or something of that sort.

Dr M'Gibbon, continuing, said he was interested to hear that the President had performed this operation so extensively; that he considered extra-abdominal treatment of the stump should be the indication in certain infected cases; and that he also hoped to perform a fourth and fifth Cæsarean section on the patient whom he had referred to. He was exceedingly obliged for the extremely kind way in which the Society had received his paper.

Meeting VIII.—July 12, 1911.

Dr F. W. N. Haultain, *President, in the Chair.*

I. *The President* showed—HYPERTROPHIED CERVIX, removed from a patient, æt. 35, whose youngest child was 10 years of age. The posterior lip of the cervix protruded from the vulva, and there was also a large ulceration on it. This had protruded from the cervix for eight years, causing a very considerable amount of discomfort; but the patient was in India, and did not want to have the operation performed until she came home. The posterior lip was exceedingly œdematous.

II. *Dr W. Fordyce* showed—FIBROID TUMOURS OF UTERUS REMOVED FOR SPECIAL SYMPTOMS AFTER THE MENOPAUSE. (Three Specimens.)

(1) This specimen was removed from a lady who had been twenty-two years in an asylum, and still was in an asylum. Attention was drawn to the tumour by the very fœtid, offensive discharge, and it was detected by an examination under chloroform. Bladder symptoms were also very troublesome—incontinence of urine getting worse and necessitating the passage of a catheter; these symptoms were accounted for by the large mass in the pelvis pressing down on the bladder. The tumour was with considerable difficulty removed; it appeared to have undergone a mucoid degeneration, and was covered over with a thick mass of jelly-like material. After the operation both the doctor and nurses volunteered the statement that the patient's mental condition was very distinctly better, and they hoped she would improve still further. But, as he (Dr Fordyce) expected, the medical officer (Dr Brown) had written to say that the patient had relapsed.

(2) The second specimen was a large fibroid removed from a single woman, æt. 49, who suffered very severely from menorrhagia. The tumour developed very rapidly, and the fact that it was still increasing in size decided the patient to have an operation, which she had previously refused. The tumour when removed, was evidently a fibroid tumour undergoing mucoid degeneration.

(3) The third specimen, and perhaps the most interesting, was from a lady who was supposed to have a cancer of the cervix which had been present, it was supposed, for a year. She then came under the care of another doctor, who asked Dr Fordyce to see her. There was no doubt that there was either cancer or something else of that nature, for the smell was so bad. The appearance of the patient, and the smell in the room made one expect cancer. In the first place, an abdominal tumour was found. The patient was bright and well-nourished. The whole vagina was occupied by this large sloughing mass—the specimen had shrunk very considerably since removal. The patient had a temperature of 103°, and was evidently suffering from septic absorption. She was brought to Edinburgh, and under chloroform it was hoped that the tumour might be removed, as it might prove to be polypoidal. On twisting the mass round, however, it seemed to have no pedicle at all. The abdomen was opened, and on going in from above, after tying the ovarian arteries, one found it impossible to get down to the uterine arteries. One did not know very well what to do, but on making an incision through the anterior part of the uterus, and through the cervix, one was able to get at the uterine arteries. After ligaturing the uterine arteries there was difficulty in getting the mass pulled out; it required to be pulled forcibly, and came out like a cork out of a bottle. One did not feel very comfortable about the result with this large putrid mass present, but apparently because the patient had had a high temperature she

had, to a certain extent, become immune to further inoculation. With the exception of slight suppuration of the abdominal wall, she made an absolutely perfect recovery and was practically well. On opening the tumour and making a section, one was astonished that the patient could have had such a condition going on without much more serious symptoms; there was a green, putrid mass in the interior of the uterus. At the time it was debated whether it would not have been better and safer to have cut this off, but in trying to twist it off the hæmorrhage was very great.

Dr Haultain said the bad, fœtid discharge for such a long time was an interesting point.

Dr Fordyce said the fœtid discharge had not continued long; she had menorrhagia for eight or ten days. There certainly was some absorption, for she had had a temperature of 103°.

Dr Haultain said the more stinking the discharge was the more innocuous it proved to be. An interesting point was the occurrence of such a condition after the menopause.

Dr Fordyce said in the first case the patient was 52; in the second 49; and in the third, although the patient was only 46, she said she had not menstruated for a year, and then began again; she had had severe hæmorrhage for about a year and the fœtid discharge for the last fortnight. How long the other patients were past the menopause he did not know, but would be able to find out.

Dr Haultain said that was the most interesting point.

III. *Mr E. Scott Carmichael* showed—UTERUS REMOVED BY WERTHEIM'S METHOD FOR MALIGNANT DISEASE OF CERVIX. The patient, æt. 50, had had all the usual symptoms for some months, and came under observation at a rather advanced stage of the disease for the purpose of operation. The uterus was quite mobile except that it was perhaps

fixed a little to the left. Only a very slight ulceration could be seen and nothing else. The operation was done on Monday. The uterus was exposed; the bladder was pushed away from the anterior vaginal wall until it was possible to clamp the vagina below the uterus. The left ureter passed through a mass of cancerous tissue, and it was necessary to divide it. A small hole was made on separating the bladder. There were also some glands between the external and internal iliac fossa, which were secondarily involved. One felt how satisfactory this operation would be if one could only get the patients early. This seemed to be the only scientific operation; one could hardly see how a vaginal operation could be really satisfactory. The patient so far was making a fight for it, and one hoped against one's own opinion that she would recover, but it would be a hard fight. He had had the kind advice of Dr Haultain at the operation.

IV. CERVICAL POLYPI EXHIBITING DECIDUAL CHANGES. (With Lantern Demonstration.)

By James Haig Ferguson, M.D., F.R.C.S., F.R.C.P., and James Young, M.D., F.R.C.S.

The following case is of double interest. In the first place, it concerns a patient from whom comparatively large cervical polypi were removed at the third month of pregnancy; and, in the second place, it is of interest from the fact that the cervical tissue entering into the structure of the polypus had undergone a decidual change. The clinical particulars are as follows:—

The patient was 49 years old. There was a miscarriage ten or eleven years ago, at the second or third month. She had

borne two full-time children—nine years and seven years ago. In the menstrual history there is nothing to note.

From 2nd to 6th October 1910 she had an attack simulating, according to the doctor in attendance, subacute appendicitis. This passed off with rest, dieting, enemata and hot fomentations. Late in November of the same year there was severe constipation. This led to a vaginal examination, when the uterus was found to be retroverted. It was replaced, and a Hodge pessary was inserted. At this time there was no evidence of anything abnormal projecting from the cervix.

For a fortnight or so she expressed herself as feeling practically well. She then, according to her doctor, "began to develop the signs and symptoms which have always accompanied her pregnancies: constant nausea, great thirst, marked constipation, and a tendency to the blocked nasal passages always noticeable when she was pregnant." The last menstruation was at the beginning of November.

Early in February she complained of a slight bloody discharge, which continued for two days. On examination two soft polypoidal masses, together about the size of the little finger, were found projecting from the external os. These were removed by Dr Haig Ferguson on 12th February. At the time all the signs of pregnancy between the third and fourth month were noted.

On 10th August 1911 the patient was safely delivered of a full-time child. The placenta was normal.

Pathological Examination.—Two different parts of the polypoidal mass were examined. They were found to possess characters somewhat resembling those presented by the ordinary polypi, which develop from the glandular mucous membrane of the cervix.

The main mass of the polypi consists of an œdematous stroma composed of a connective tissue of a low or embryonic type (*i.e.*, there is little attempt at differentiation). Embedded

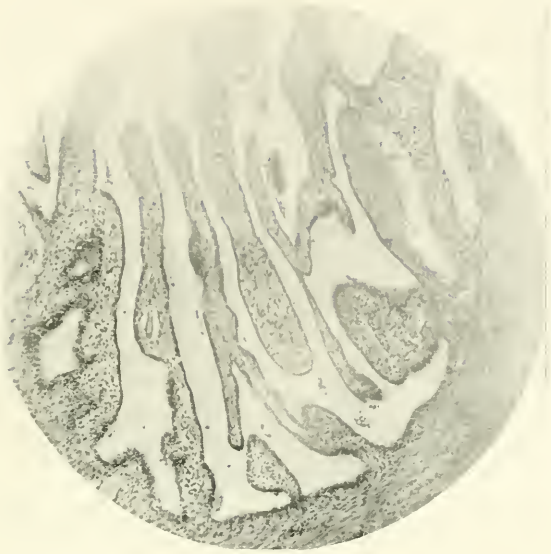

FIG. 1.

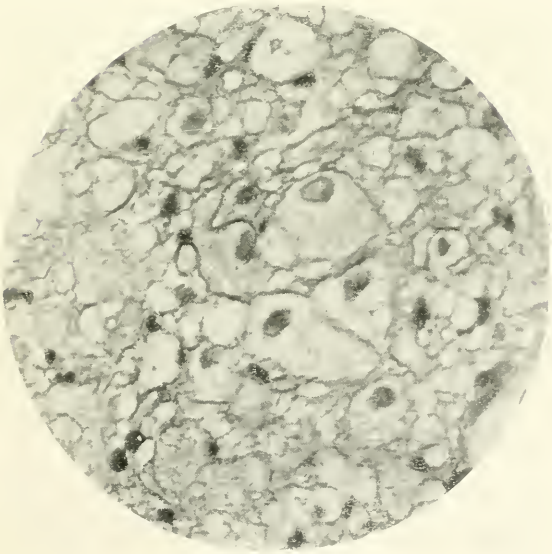

FIG. 2.

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in this there are glands lined with the single layer of palisade epithelium characteristically present in the racemose glands of the lining of the cervical canal.

There are some points of especial interest in connection with the *connective-tissue stroma*. As mentioned, it presents little differentiation of structure, the cells unite freely with one another, and there is an absence of the distinct fibrous appearance present in the mucosa of the normal cervix. The conditions suggest a young, newly-formed tissue, and, in many respects, coincide with those seen in the stroma of the corporeal mucosa (see Plate, Fig. 2).

Cause of the Œdema and Blood Escape.—There is a marked œdematous collection throughout the entire connective tissue. The vessels are, in many places, distended, and in some regions there are small areas of hæmorrhage, some of which are large. In addition there are small areas which stain deeply with eosin and in which the nuclei are disintegrating or have actually disappeared. These areas are scattered throughout the tissue and correspond to commencing necrosis, doubtless dependent on a local interference with the blood-supply of the polypi.

On careful examination it is found that the watery escape into the tissues is associated with changes of considerable importance. It is found that, in the process, the cells have been spread apart, and the appearances would at first sight suggest that there has been mechanical expansion of the intercellular spaces dependent on a fluid escape in response to an increase in the intravascular tension.

On examination under a high-power lens it is found, on the other hand, that the appearances all prove that *we are dealing with a protoplasmic change that has resulted in an imbibition of fluid*. We can make out that the intercellular spaces correspond to completely closed-in fluid spaces, and throughout we can see dropsical accumulations in the proto-

plasm, which has become swollen and split up into fluid chambers separated by fine cytoplasmic films (see Plate, Fig. 2). The appearances are amenable only to the explanation given above.

The changes just noted in these polypi have been described in full by one of us as a constant alteration in the uterine mucosa in menstruation and in early pregnancy, in both of which conditions we have a diffuse protoplasmic change tending to lead to an imbibition or dragging of the blood from the vessels. It is changes of this kind that are responsible for the alterations in the endometrium by which the early ovum is furnished with its blood supply.[1] Space will not permit a fuller account of the significance of these tissue changes.

Decidual Change in Stroma.—In places the stroma cells are enlarged in a truly decidual fashion (see Plate, Fig. 2). They may be lying singly, in small groups or in dense masses resembling in every respect the appearances present in the decidua compacta of normal pregnancy. This change is of especial interest, as it is occurring in this specimen in tissue derived from the stroma of the cervical mucosa, in which a decidual alteration is usually said to be absent during pregnancy. We believe that it is possible to explain both of these changes, the ease with which the œdema has taken place and the decidual alteration in cervical tissue, as due to the fact that we are dealing with young, newly-formed tissue exhibiting characters which identifies it closely with the endometrial stroma, in which the changes readily occur during pregnancy in response to the materials elaborated by the chorionic cells.

The microscopic appearances suggest that the polypi are of recent growth. From the history it will be noted that

[1] Young (1), see pp. 61-79 of this volume. (2) "The Uterine Mucosa in Menstruation and Pregnancy; the Action of the Chorionic Cells and the Function of the Decidua," *Proc. Royal Soc. Med.*, June 1911. (3) *Reproduction in the Human Female*, Messrs William Green & Sons, Edin. and Lond., 1911.

there was no evidence of their existence early in pregnancy, and, whilst it is impossible to say that they had developed *de novo* during the pregnancy, there is every reason for the belief that the pregnancy had at any rate stimulated their growth.

The Glands and the Epithelium.—As we have described above, the epithelium of the gland spaces and on the surface is of the cervical type. The glands are clearly undergoing an increase in their numbers, and in places are distended in a cyst-like fashion, the cavity being occupied with a homogeneous faintly-staining secretion. There is, however, an absence of the marked glandular increase associated with the typical cervical polypus. Another point of interest is found in the fact that the surface of the tumours has in places assumed a definite papillomatous change. In Fig. 1 is seen a section through the surface level which has cut across the outwardly projecting stalks, consisting of stroma core covered by epithelium.

Dr Berry Hart asked if the cells which were described as decidual were not really dropsical cells.

Dr Young said the changes were not those found in dropsical cells. Whilst there was, as he had pointed out, a diffuse œdema, the cells in question were clearly decidual. They were enlarged to a size never found in other conditions, and the cell was seen to absorb the stain uniformly. In addition, the nucleus was central. In dropsical cells it was displaced to the periphery.

V. SIMPLE PAPILLOMATA REMOVED FROM VAGINAL VAULT AFTER VAGINAL HYSTERECTOMY FOR CERVICAL CANCER. (With Lantern Demonstration.)

By James Haig Ferguson, M.D., F.R.C.S., F.R.C.P., and James Young, M.D., F.R.C.S.

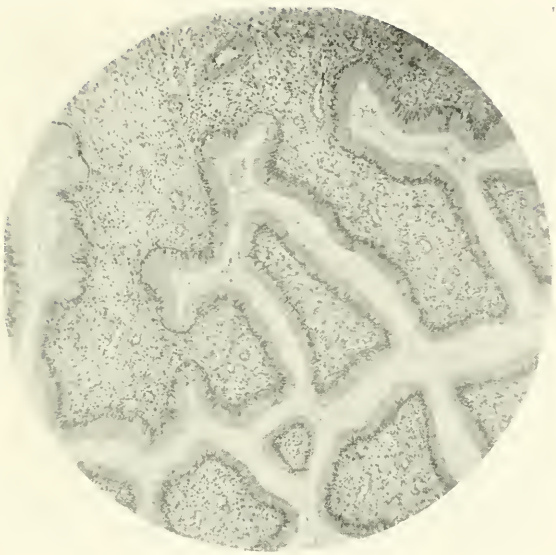
The following are the notes of a patient under treatment in Ward 36, Royal Infirmary, Edinburgh:—

Mrs S., æt. 47; she had seven children, the youngest being 6 years old. On 27th March 1911 a vaginal hysterectomy for squamous epithelioma of the cervix was carried out by Dr Haig Ferguson. At the time it was doubtful whether all the disease was being removed. On 24th April 1911 two soft, bleeding projections were removed from the vaginal wall at the site of the wound in the vault, which was completely healed.

The pathological report of these outgrowths is of considerable interest. Microscopic examination revealed in them characters somewhat similar to those found in a villous papilloma.

At no part was there any evidence of the ordinary squamous epithelium of the vagina. The connective tissue is markedly œdematous, and in places is infiltrated with blood. Many of the vessels are greatly expanded. In places there are large tracts of degeneration. The surface is in parts devoid of an epithelial covering. In most places, however, this is present, and consists of an epithelial layer of flat or cubical cells arranged in a single row. In other regions the cells are several layers deep.

The surface of the growths exhibits appearances of great interest in the existence of distinct villous outgrowths of the



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connective tissue covered by epithelium. The connective-tissue cores are œdematous and in places hæmorrhagic. There is in parts great distension of the vessels. In some places they are disintegrating in a manner similar to the change encountered in the main mass of the growth. The epithelium is one, two, or more layers deep. In places it is absent.

The projections vary greatly in appearance and size. Sometimes they are broad, at other times they are long and filamentous. After arising by a broad stalk they may break up into fine processes. They are often seen to branch. These appearances are shown in the Plate. In some regions the epithelial increase is associated with a projection of epithelium-lined spaces into the superficial parts of the main growth.

The appearances, which have just been described, are somewhat puzzling. It is difficult to give an explanation of the exact origin of the papillomatous growth in this unusual situation. At first sight it might seem that one was dealing with the epithelial lining of the tube, which had been cut across, and the divided end of which was pouting into the vaginal vault. The situation in relation to the scar might be supposed to lend support to this conception. There is no evidence, however, of a lumen in the outgrowths, and the arrangement of the epithelium is such as to negative such a simple explanation. In the first place, the epithelium-covered processes develop over the surface of the growths in a very irregular fashion. Some parts are flat, whereas others are beset with an intricate system of villous growths. In the second place, where the outgrowths occur they do not correspond to the simple arrangement of the mucous folds such as is found in the inner portion of the tube, *i.e.*, the end with which one would be dealing, as the tube is cut across close to the lateral margin of the uterus. On the other hand, it may possibly be the case that in the outgrowth we see an actual new formation derived from the tubal mucosa, which has

assumed a papillomatous state. As has been noted, however, the epithelium which covers the surface of the main growths and the villous projections is different from that found in the tubal mucosa. Instead of being columnar and ciliated it is, for the most part, flattened or cubical. This, however, may simply represent an incidental alteration.

The other explanation which may be advanced is that the papillomata are derived from the vaginal mucosa, though, as has been mentioned, there is at no part of the growths any trace of the squamous epithelium of the vagina.

On the whole, it would seem that we are dealing with papillomata, simple in nature, derived from a local overgrowth of the mucosa of the inner ends of the tubes, where they have been cut across in the process of detachment of the uterus.

The practical point brought out by this case is that bleeding after a vaginal hysterectomy may occur even after a complete healing of the vaginal vault as the result of a constant oozing from such structures. We have referred to the congestion, œdema, and hæmorrhage exhibited by them.

VI. A CONTRIBUTION TO THE THEORY OF ENZYGOTIC (MONOCHORIONIC) TWINS.

By D. Berry Hart, M.D., etc., Lecturer on Midwifery and Diseases of Women, Surgeons' Hall, Edinburgh.

Introductory.—In the course of an inquiry into the possible autonomous unit-characters making up the human frame, the case of enzygotic twins came up, and as it forms a distinct and interesting section of this general question, it lends itself to separate consideration. Another reason is that Dr Ballantyne has often brought the occasional deformity of such twins before us, without, I am afraid, receiving as much encouragement as

he should have. I am, however, specially indebted to him for his many valuable communications, as without these it would have been impossible for me to have taken up this subject.

Two of the most interesting kinds of enzygotic twins are those of identical twins and the allantoido-angiopagus forms.[1] The latter usually consist of a normal twin and a deformed one, the deformities being very frequently those of exact defect. Such twins are derived from one fertilised ovum (zygote), and are therefore monochorionic or enzygotic (one-zygotic), as I have termed them. They are always of the same sex; the umbilical cords communicate near the placenta, and the sexual organs are usually normally developed and always similar. We may also have twins which are completely separated from one another except that the cords may unite, or those whose bodies are more or less united.

The classification given is as follows:—B. Polysomatous (two, or more, individuals involved), but monochorionic. I. Twins, entirely separate. II. Twins, united only by the vessels of their umbilical cords (omphalo- or allantoido-angiopagus). (*a*) Paracephalic; (*b*) acephalic; (*c*) amorphous. III. Twins, united more or less completely (double monsters). (*a*) Symmetrically united—(1) Syncephalic; (2) dicephalic; (3) thoracopagous. (*b*) Asymmetrically united—(1) Cephalo-parasitic; (2) prosopo-parasitic; (3) trachelo-parasitic; (4) thoraco-parasitic; (5) gastro-parasitic; (6) lecano-parasitic; (7) melomelic (slightly modified from Ballantyne).

Explanatory.—The human zygote (*i.e.*, fertilised ovum) usually develops into a single fœtus. In a certain proportion of cases, however, two perfectly normal fœtuses are formed from the one zygote, and these may be exactly similar, and are then termed "identical or fraternal twins."[2] These are always

[1] Twins with the umbilical cords communicating. I shall discuss the correctness of the term later.

[2] This is used as equivalent to male and female, like the German Geschwister.

of the same sex, have been completely separated, and may be so alike in every way in adult life as to be often mistaken for one another even by those who are familiar with them. Identical triplets and even quintuplets may occur.[1]

In the allantoida-angiopagus twins we have a similar condition of identity, but one only is perfect, or nearly so, the other may be, by defect, so deformed as well to deserve the term monster. Thus the head may be awanting; the trunk, on the other hand, may be absent or very defective; and finally only the head may be present. The internal viscera may be defective or awanting, and the defects may be so serious that the fœtus can only exist as an apparent parasite.

The two fœtuses may, however, be more or less joined, and thus we have the well-known double monstrosities given at III. in the previous table of classification. In such the sexes are also always similar, and the internal genitals normally developed. Certain peculiarities are present in the intestinal tract, as in cases recorded by Berry and Kamann.

In considering these remarkable and, at first sight, seemingly inexplicable anomalies, one must start with identical-separate twins, and endeavour to determine exactly, so far as we can, *what happens when normal and healthy identical twins are formed.*

Hitherto this question has usually been answered by the words, "an equal primary cleavage division gives rise to two halves of the zygote, and each of these develops as a fœtus." This, however, does not lead us very far, and the statement has been disputed, as we shall see.

In the modern conception of Evolution we owe to Weismann we look on a zygote in its somatic part[2] as containing determinants which are the elements in the zygote causal to the future developed organs—the determinates—of the

[1] See Wilder, *Amer. Journ. of Anat.*, iii. 395.

[2] That part which will develop the "soma" or body.

adult, and we may say, no determinants, no determinates. These terms form a merely hypothetical causal terminology not demonstrable, and the real nature of the determinants is unknown.

Following Mendel we can further regard these determinants as causal to the adult autonomous unit-characters, and that in transmission they do not blend but interchange in heredity in a definite manner. In the plant-crossings Mendel and many others have practised, contrasted unit-characters have been shown to sift out unchanged in subsequent generations in definite ratios, and the point I have urged in this is that this is due to a ratio-arrangement *in the determinants* of the propagative part of the zygote, following the law of probability. This conception of the ultimate results being determined by what happens in this part of the zygote gives us an extension in Mendelism of the greatest value, and to understand the ultimate results in identical twins we must think in terms of the determinants.

When identical twins are formed there is either a halving of *all* the determinants (in the somatic part of the zygote), or a doubling of them *all*, the latter being more feasible, and then an equal segregation of them into two groups, thus making a complete division of the zygote determinants. In this way twins arise that are absolutely identical, even to the finger-prints, as Wilder has shown. Finger-prints are never identical, in the single individuals of the human race, as no instance of such identity is known. Wilder's paper on this subject is most interesting.

We can think of reproduction in the amœba in the same way. Thus when a bud is formed we may consider that the amœba determinants have doubled and then segregated into two groups, and this is an analogy familiar to us all. Any incomplete doubling in this may cause a variation.

This description of doubling and segregation[1] involves that of equal division (often expressed as symmetrical cleavage of the zygote), and the ultimate results, complete equivalence and separation in the fœtus and adult, come out thus as the result of zygotic changes. Equal cleavage as the expression of division is therefore unnecessary. When the zygote is formed there is a multiplication of the determinants so as to produce the primitive germ cells, and this may be repeated in single zygotes forming identical twins, and would give a feasible explanation of polydactyly and the rare case where five hearts existed in a single hen (see Schwalbe).

In identical twins the segregation of the doubled determinant groups is complete. Each fœtus is equivalent and has its own umbilical cord, the cord being the posterior end of the embryo, as His long ago correctly pointed out.

When, however, all the determinants are not doubled but only certain of them, half of those that are doubled segregate to one side, and form an incomplete set, while the remaining set is necessarily complete, inasmuch as it has the determinants that are not doubled and its equal share of those doubled. Thus we get the paracephalic, acephalic, and amorphous varieties according to the amount of deficiency in doubling.

When there is union of the twins, more or less, the cause is incomplete segregation, comprehending also incomplete division, and thus the varieties given at p 235. The umbilical cord anomalies are likewise due to this incomplete segregation, inasmuch as the cord is part of the embryo.

Thus identical and allantoido-angiopagus twins may be considered as being due to complete or incomplete doubling, as the case may be, followed by complete segregation so far as the multiplied elements are concerned. If there is exact and complete doubling and complete segregation we get identical twins;

[1] This term is used in the ordinary sense of separation into a definite group.

incomplete doubling and segregation with consequently incomplete allotment gives us the many varieties classed as allantoido-angiopagus monsters, or united twins.

I must now consider some of the other views brought forward in explanation of the remarkable deformities occurring in enzygotic twins.

The communicating umbilical systems and the acardiac condition so often present seem to me part of the process and not to be causal. In the acardiac condition the cardiac determinants have not doubled, and therefore the healthy twin alone possesses a heart. The incomplete twin will necessarily have a smaller share of chorion and of blood-vessels, but this is a result, not a cause. All explanations based on the later growth of the allantois in one twin are open to the objection that the allantois plays a rudimentary *rôle* in the human species and is phylogenetic in significance. Twin embryos have also been found in the earliest stages before any allantois forms. The placenta is really an organ of the chorion, not of the allantois.

The explanation I bring forward is a simple one, and explains the curious result in allantoido-angiopagus twins in an understandable way. It is only part of the rich heritage the work of Weismann and Mendel is giving to obstetrics.

It seems to me, finally on this head, important to note that the explanations hitherto given of allantoido-angiopagus twins have assumed that they were due to changes in the formed fœtus. The explanation I have urged is based first on Weismann's conception that what is the result in the developed organism is due to causal elements in the zygote. To Mendel's discovery that the organism is built up of autonomous unit-characters I add the view that the ultimate results in the adult organism are due to changes which have taken place in the zygote and the gametes forming it. This seems to me to introduce into our views of the formation of such monstrosities

associated with twinning a simplicity and accuracy hitherto awanting.

The dissection of specimens of conjoined twins has yielded valuable results in the hands of Kamann, R. J. A. Berry, and many others. Professor Berry has drawn especial attention to the absence of the gall-bladder in one of the twins, and naturally says that it is inexplicable if twins result from a simple cleavage. He urges the following as his explanation:—

"It is safe to assume that the two embryos in each case have had only one yolk-sac between them; that is to say, they have been developed from one ovum.

"Commencing upon this hypothesis, and remembering the lateral nature of the fusion of the twins, I first assumed that two primitive grooves had been lying side by side upon a single ovum. I endeavoured to account for these two primitive grooves by assuming bilateral segmentation of the ovum, but I was forced to abandon this position as untenable, inasmuch as if worked out it will be found to necessitate the formation of two yolk-sacs and two amnions, and their subsequent conversion into single structures by fusion. Quite apart from these objections there are others of an even more insuperable character, and consequently this supposition of bilateral segmentation of a single ovum was subsequently abandoned in favour of the following theory, which appears to offer a more satisfactory explanation of the phenomena.

"1. *The Explanation of the Lateral Fusion and the Common Epiblastic Covering of the Fused Areas.*—This explanation is based on the supposition that the two embryos have been developed from a single ovum *without* bilateral segmentation. The two primitive grooves have lain side by side in such a way that the caudal ends of the embryos have been rather nearer to each other than the cephalic ends, or, at all events, the central portions of the primitive grooves have either been nearer each other than any other portions or have been in actual contact.

What I then suppose to have taken place is illustrated in Fig. 2,[1] and granting the original hypothesis—two embryos developed from a single ovum without bilateral segmentation—an easy explanation results as to the lateral fusion of the twins, even to the greater fusion in the second example than the first, and also as to the common epiblastic covering of the fused areas and the single jejunum tube. All are easy of explanation on this hypothesis, and difficult, if not actually impossible, upon any other.

"2. *The Explanation of the Single Duodenal and Jejunal Tubes and the Single Liver.*—The hypothesis adopted in the preceding paragraph assumes the presence of a single yolk-sac common to both embryos, and Fig. 3[1] illustrates my conception of the evolution of the single duodenal and jejunal tubes from this common yolk-sac. Both the pyloric ends of the duodenal tube have undoubtedly been developed from independent foreguts, whilst the intermediate portion, with the biliary duct and the single jejunum, have, in my opinion, been developed from the common yolk-sac or midgut. I say 'in my opinion,' because such an assumption necessitates the liver being a midgut development, and the numerous works upon embryology which have been consulted appear to me to leave the question an open one. Keith, in his *Human Embryology and Morphology*, states that the foregut becomes the pharynx, œsophagus, stomach, and that part of the duodenum situated in front of the opening of the common bile duct; that is to say, he appears to regard the liver as a midgut development. With this I entirely agree, inasmuch as in both these cases there is but one common bile duct, which opens into the intermediate portion of the single duodenal tube. Thus the liver is proved to be a single structure common to both twins, and not two liver buds fused together, and as an additional argument in support of this view it may be stated that each

[1] See Berry's paper, *Edin. Obst. Trans.*, vol. xxviii.

liver has but one Spigelian lobe. For these various reasons I am therefore of opinion that these cases prove the liver to be a midgut development, and that Keith is perfectly correct in his views upon this point.

"The hypothesis upon which the lateral fusion was explained suffices, therefore, for the single duodenal and jejunal tubes as well as for the single liver.

"3. *The Suppression of the Gall-Bladder in the One Case and its Presence in the Other.*—As the gall-bladder is known to be developed normally as a diverticulum from the common hepatic stalk in the second month, it is perfectly easy to understand the suppression of this viscus in the one case and its presence in the other; it is at the same time impossible to offer any theory as to *why* the gall-bladder diverticulum should thus grow out in the one case and not in the other.

"That twin embryos do develop side by side upon a single ovum is proved by the remarkable case published by Miss Duncan in the *Proceedings of the Anatomical and Anthropological Society of Aberdeen*, 1900-1902, under the title of 'The Anatomy of a Double Chick Embryo,' and the cases with which this paper deals would further seem to prove that bilateral segmentation of an ovum is unnecessary for the evolution of twins. The condition of the alimentary canal proves conclusively that the twins in each case have possessed but one common yolk-sac; bilateral segmentation of a single ovum would result in two yolk-sacs, and hence these cases seem to afford indubitable proof of the truth of the hypothesis adopted herein, namely, double embryos on a single ovum without bilateral segmentation. Further proof of the truth of this theory is afforded by Kamann's 'Zwei Fälle von Thoracopagus tetrabrachius,' published in the *Archiv für Gynäkologie*, Bd. lxviii. H. 3, where the condition of the alimentary canal in each case was identical with that recorded in the present paper. The present writer's (Berry's) theory, therefore, not only

explains the two cases now under discussion, but also suffices for Miss Duncan's case and for those of Kamann. If these several cases and the resulting facts may be accepted as proving the writer's contention, the labour and thought bestowed upon these embryological problems will not have been spent in vain, for two important scientific results follow:—(1) Bilateral segmentation of a single ovum is unnecessary for the production of twins; (2) the liver is a midgut development" (*Edin. Obstet. Trans.*, vol. xxviii.).

This does not, however, explain how the two primitive grooves arose. If not by bilateral segmentation of the ovum, then how? If one assumes that there are two primitive grooves to start with, no further explanation is necessary.

Any idea of polyspermy cannot in my opinion be entertained. Each spermatozoon would have half its chromosomes when matured to a gamete, while the ovum would have only its half sufficient for one gamete, and, as pointed out by Turner, identical triplets would require three male gametes and one female one. According to my theory, there would be in identical triplets tripling of the determinants and a complete and equal segregation of the three groups.

The explanation I offer is as follows:—The twins Professor Berry describes arose from one zygote. There was doubling of the determinants, except in the case of the gall-bladder, with incomplete segregation and therefore fusion in the lateral line. The primitive gut was thus a common one, as the internal somatopleuric folds could not curve in, and only the outer one in each fœtus did so. The amnion is a single one, because the solid plugs from which the amnia arose were double and fused.

The yolk-sac is a diverticulum from the common primitive gut, and therefore single.

The explanation Dr Berry gives as to the liver and circulatory conditions is excellent.

The view I have advanced explains, I think, Professor

Berry's difficulty. The gall-bladder is single because its determinants were not doubled. Bilateral segmentation is not necessary for the production of twins; it is not even necessary for their separation. *It is the doubling of determinants in the zygote and their segregation that are the essential things*, and this involves the cleavage idea hitherto always used.

I may now sum up the process in each special case; each of course is formed from a single zygote.

Identical and Separated Twins.—Equal doubling of determinants; complete segregation.

Paracephalic.—Doubling of cephalic determinants; incomplete doubling of others; segregation of half of doubled determinants, which thus form the paracephalic monster.

Acephalic.—No doubling of cephalic determinants but of the others, and thus deformed twin acephalic. The other twin possesses, of course, a head.

Where the monster twin is represented only by a head, leg, or arm the determinants of these alone were doubled and segregated.

In a previous paper in the *Edin. Obstet. Trans.*, 1910-11, I have endeavoured to show that in deformities in single fœtuses the defect is due to loss of determinants when the ovum matures, *i.e.* when the polar bodies are thrown off. This holds good for single deformities not associated with twinning, and need not be further alluded to.

I now wish finally to consider the question of nomenclature.

The term allantoido-angiopagus does not seem to me a very fortunate one. In the first place the allantois is a rudimentary structure in the human fœtus, and in addition the irregular distribution of vessels is a result, not a cause, of the condition.

The essential point about such twins is that they are developed from one zygote, and are thus termed enzygotic. The subjoined table gives the possible result in such cases when twinning is a result.

Enzygotic or Monochorionic Twins.

I. Complete and exact doubling or tripling of determinants; complete and equal segregation. *Identical twins or identical triplets.*

II. Incomplete doubling and complete segregation, part of funis excepted. Imperfect twin — Paracephalic, acephalic, amorphous.

III. Incomplete doubling and incomplete asymmetrical segregation — Cephalo-parasitic, prosopo-parasitic, trachelo-parasitic, thoraco-parasitic, gastro-parasitic, lecano-parasitic, melomelic.

IV. Complete doubling and symmetrical incomplete segregation—syncephalic, dicephalic, thoracopagus.

Literature.

This is fully given in Schwalbe's *Missbildungen* and Ballantyne's *Antenatal Pathology.*

The following extracts from de Vries' *Intracellular Pangenesis* (Chicago, 1910) are of value:—"We must therefore assume that they" (the pangens, Weismann's determinants) "assimilate and take nourishment, and thereby grow, and then multiply by division, two new pangens like the original one usually originating at one cleavage" (p. 70, Gager's translation).

"This process of dispermia" (fertilisation of one ovum by two sperms, Boveri) "leads to . . . a triple number of chromosomes, . . . and then . . . the germs . . . show deviations from the normal type" (do not form identical organisms) (*op. cit.*, p. 235).

VII. A MONGOL OF A PRIMIPAROUS MOTHER: PARENTS BOTH YOUNG AND HEALTHY: TRAUMATIC SHOCK TO MOTHER EARLY IN THE PREGNANCY.

By Henry M. Church, B.Sc., M.D.

A mongol may be defined as a product of exhaustion. But it is not always so. She is, for it is more frequently a girl, a child with antenatally arrested physical and mental development, with brachycephaly as a prominent feature, a circumstance which sometimes renders delivery difficult. Like other practitioners I have had a small percentage of mongols, and these have been children coming late in the family when the mother was about the climacteric period, or, it may be, exhausted with child-bearing. The mothers in some recorded cases have been subject to some infectious disease during pregnancy, or suffered from a wearing-out mental or bodily strain, such as anxious nursing.

Dr Bertram Hill says: "In my own series of 8 cases there was a definite history of bad health in the mother during pregnancy in 4: in 2 of these 4 the mother gave spontaneously an account of fright whilst carrying the child." [1]

Weygandt, in a study of 37 cases, states that "in the majority of cases the patients were last children of a large family, and that the parents were often old, with disparity in their years." The mongol to whose case I refer in the paper is in these points exceptional. Her parents were young and healthy. She is now a girl of 10, was born at the full time, and weighed nearly 8 pounds. Delivery with forceps was a matter of difficulty, on account of a narrow pelvis and the rounded brachycephalic head of the child.

[1] *Quarterly Journal of Medicine*, October 1906.

The mother was a strong woman, with good breasts, which enabled her to nurse for some time. Unfortunately her lactation was continued for three months after she was again pregnant. This overlapping was bad for the suckling, but could have nothing to do with the evolution of its mongol type. Three healthy boys have been born to the parents since.

The expression of the child has all along been more or less vivacious and mobile. She is observant and imitative, and very different from the dull, apathetic expression of the cretin, but is incapable of being taught like children of her own age. She is left-handed. In the hope of improving the mental condition, or at any rate the general physical condition, enlarged tonsils and adenoids were removed at 4 years of age, with the desired result.

Syphilis, parental alcoholism, or tuberculosis do not seem to enter markedly into the causation of a mongolian birth. In my case the only taint discoverable is that a paternal great-grand-aunt suffered from lupus of the face. This, however, may be or may not be a sidelight of pathological import. With the exception of dwarfed nasal bones, large fontanelles, and an unusually high, narrow palate there may be said to have been nothing of the nature of congenital malformation. Hill points out that malformations occur more frequently in the mongolian than in any other variety of idiocy, and probably indicate some influence acting very early in intra-uterine life, about the second month, and may be synchronous with cerebral changes.[1]

Apropos of this, the important point of my communication is the fact that the mother, about ten days after conception, met with a serious fall from her bicycle. And though, at such an early date, there has not been made out by embryologists any structures referable to brain, it is undoubtedly true, as in the case of all maternal impressions, that any undue

[1] *Quarterly Journal of Medicine*, October 1908.

influence upon the mother's nerve-centres often profoundly affects the fœtus. But how is not known. Subtle influences, much less important than a cycle accident, gives rise to altered circulation in the brain and nerve-centres of the mother and influence the new life at its very springs. Without appealing to the observations of modern science on these biological phenomena in man or in animals, let us recall in this connection how subtly Jacob influenced his flock, by impressions made to the eye, at the time of conception at the watering-troughs.

"Violent moral emotions," says Paul Sallier, "in the mother during pregnancy have an indubitable action upon the evolution of the fœtus, and this action is all the more marked as the motion is more violent, and especially when it is produced at a period but little removed from the time of conception."[1] The other mongolian cases I have met with have been associated with infection, or intestinal auto-intoxication in the mother, which seems to have brought about irremedial changes in the nervous system of the fœtus. Direct traumatism, as in the case before us, or anything that will cause circulatory disturbance in the mother, appear to imperil those nerve-centres in the fœtus which direct its normal growth and development. Innumerable little accidents bring on abortions, and if even no abortion follow such accident, a lasting and incalculable impression may have been made on the development of the plastic and growing embyro. The subject is one of far-reaching importance, and bears upon the problems of arrested developments and other antenatal pathology.

The conclusions from this short record are (1) that, though mongolian offspring is generally from an exhausted mother, or from parents in whom there is disparity of years, a mongol may occur in a primiparous young mother who has been subjected to a traumatic physical shock early in her pregnancy.

[1] *Twentieth Century Practice of Medicine*, vol. xii., p. 267.

(2) The importance of protecting an expectant mother from all bodily and mental shocks.

Dr Berry Hart said they were indebted to Dr Church for his careful paper. No definite idea of the pathology of the mongol idiot could really be formed. These patients were all apparently exactly alike. Dr Church gave a traumatic explanation which was one of the best which could be brought forward. In all these cases he (Dr Berry Hart) would inquire into the history very minutely and see if there was not any nervous affection. It was important to get a correct family history, but it was not always easy to obtain it.

Dr Oliphant Nicholson said he had had one mongol, which came at the end of a large family. The father and mother were perfectly healthy. These children seemed in many respects to show the same characters as cretins—the same type of circulation, cold dry skin, scanty hair, etc. He had a feeling that some derangement of the ductless glands might account for the condition. He thought the want of thyroid secretion had something to do with it, because some of these cases improved under the action of thyroid extract, but not in anything like the striking way that cretins did. There was an important interdependence of all the internal secretions, and some obscure states of disease were undoubtedly produced by the upsetting of their normal relationships to one another, quite apart from the want of any one particular secretion.

Dr Fordyce was rather disappointed with Dr Berry Hart. Why not throw out some determinant to explain the mongol condition.

Dr Berry Hart said he had no "facts." He thought there might be something in the suggestion. In the matter of anencephalics, Ninian Bruce had told him that there was

a certain amount of arrest. The pituitary might have something to do with it.

Dr Ballantyne said the question of traumatism in early pregnancy had often struck him as probably of far more importance than was generally supposed. It was often grouped with mental effects occurring at the fifth or sixth month. There was something to be said for a very early physical impression. Take, for example, the hen's egg. People who are in the habit of dealing with incubators know that the eggs have to be allowed to stand for several days; if they are taken immediately off the train, abnormal chickens result. That is the so-called scientific side of the argument. At the same time in the human subject the ovum is very securely and cosily packed away, and there must be a very severe shock to cause harm. He had seen marked brain disease in an infant where the mother had a most extraordinary amount of anaemia during the pregnancy, so much so that pressure was brought to bear to try and get the child brought into the world prematurely. He had never delivered a woman with less loss of blood. For some time the child was thought to be an idiot, not exactly a mongol. Another cause of abnormality is where the infant is suckled by a mother carrying a fœtus in the uterus; often such children were malformed.

Dr Church replied that his patient was in a good position. The only history of disease that he could get was that a great-grand-aunt had had lupus. The patient had passed out of his care and observation, but he had heard that thyroid had done the child good.

VIII. TWO CASES OF PERITHELIOMA (*A*) OF THE UTERUS; (*B*) OF THE TUBE. (Lantern Demonstration.)

By A. H. F. Barbour, M.D., LL.D., F.R.C.P.E., Lecturer on Gynæcology, University of Edinburgh, Gynæcologist to Royal Infirmary, Edinburgh; and B. P. Watson, M.D., F.R.C.S.E., Lecturer in Midwifery and Gynæcology, School of Medicine of the Royal Colleges, Edinburgh.

The tumour known by the name perithelioma has only of late years been much discussed. It is so called because of the arrangement of the cells round the blood-vessels, the endothelial lining of which is intact. The cells composing the tumour are arranged outside this endothelial lining in a direction radial to the vessel. The cells are supposed to be derived from the perivascular lymphatics, and are therefore endothelial in origin, so that the tumour is really an endothelioma derived from the lymphatics.

To judge from the short descriptions in the latest text-books of pathology, the tumour is not a common one in the female pelvic organs, and this is borne out by the few cases recorded in the literature. For this reason we wish to place on record the two cases which we have been fortunate enough to obtain.

Case I. *Perithelioma of the Uterus.*—The patient was a married woman, æt. 58, who had had three children. She was admitted to Ward 35, Royal Infirmary, Edinburgh, in October 1910. Up to five months before admission she had been quite well. Menstruation ceased when she was 48. In June 1910 she began to have a white vaginal discharge, at first small in amount, but rapidly increasing and becoming fœtid in odour. A month later it became blood-stained and

the fœtor was not so marked. Just before admission it had become more watery, but was still occasionally blood-stained and fœtid. A fortnight before admission she noticed a swelling in the lower part of the abdomen.

On examining the patient this swelling was visible as a small rounded projection above the pubes and slightly to the right of the middle line. On vaginal examination the cervix was hard, not papulous, and slightly drawn up. Bimanually the swelling was found to be incorporated with the uterus, forming a uniform swelling about the size of a five months' pregnancy. It was of firm consistence and slightly tender. A diagnosis of fibroid tumour undergoing some degenerative change was made, and the abdomen was opened.

The uterus was found to be uniformly enlarged, free from adhesions, and quite movable. It was removed by amputating low down through the cervix, and the operation was completed in the ordinary way. Recovery was uneventful, and so far the patient has remained well.

Specimen.—The parts removed consist of the uterus together with the greater part of the cervix. It is enlarged to about the size of a five months' pregnancy, measuring 6 inches in vertical and 4 inches in transverse diameter, and is of a regular ovoid shape. The peritoneal covering is everywhere intact. The cervical canal admits the finger, and through it the lower pole of the tumour can be felt free inside the uterine cavity. At the line of amputation there is no trace of new growth.

On section, after hardening in formalin, the enlargement is seen to be due to the presence of a tumour growing from the fundus, the upper two-thirds of the anterior wall and upper one-third of posterior wall. The lower pole projects free into the dilated uterine cavity (Plate I., Fig. 1). The section of the tumour is of a yellowish-white colour, resembling brain tissue in appearance and consistence. Throughout there are many hæmorrhages. These are largest and most numerous towards the fundus, and in this region there is an area of necrosis where

FIG. 1.

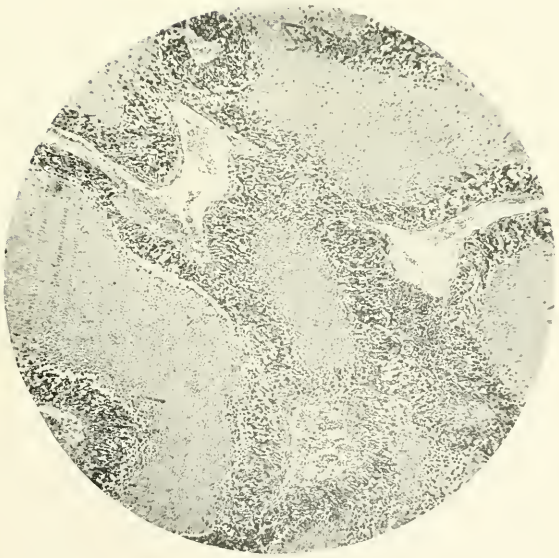


FIG. 2.

[*To face page* 252.

the tissue is breaking down. The appearances are thus very like those of an ordinary sarcoma.

Over the area of the uterine wall from which the growth is springing there is extensive invasion by the tumour, so that at the fundus the muscular wall is reduced to a mere shell. The extent of the invasion diminishes towards the lower pole of attachment. The mucous membrane and muscular coat beyond the site of the tumour are to all appearances healthy.

Microscopic Examination.—The great part of the tumour, especially towards the centre, is composed of necrotic tissue containing extravasated breaking-down blood, but otherwise devoid of definite structure. Towards the uterine wall, islands of tumour cells are present in the midst of this necrotic material. Each island consists of a blood-vessel, the endothelial coat of which is intact, but the other coats are replaced by radially arranged cells (Plate I., Fig. 2). The cells are of somewhat irregular outline; each contains a large deeply staining nucleus, and most are set at right angles to the lumen of the vessel (Plate II.). The cells at the periphery of the area are in various stages of degeneration. These appearances are constant throughout the growth, the vessels in some cases being cut longitudinally and in others transversely. In some parts there are appearances suggestive of the origin of these cells from the lymph channels, small spaces being visible with the cells grouped round them. Sections of the uterine wall at the site of the tumour show the tumour cells invading the muscular coat. Here the perivascular arrangement of the cells is not so distinct.

Case II. *Perithelioma of the Fallopian Tube.*—The patient was an unmarried, multiparous woman, æt. 53. Previous health had been quite good. Menstruation ceased at the age of 51. Seven months prior to admission she had noticed a swelling in the lower part of the abdomen towards the left side, her attention having been drawn to it by pain in that region. An

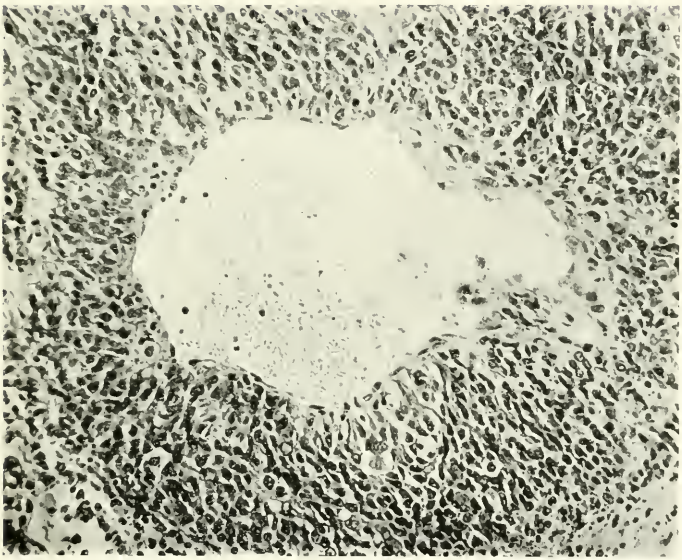
irregular vaginal discharge then made its appearance. It was sometimes pure blood, sometimes a yellow, pus-like discharge, but most often yellowish and streaked with blood. The abdominal swelling increased in size, and the general health began to deteriorate.

On admission to hospital the patient was emaciated. The abdomen was tumid, and there was a marked projection extending as high as the umbilicus and slightly to the left of the middle line. This swelling was hard and almost filled up the left iliac fossa. On vaginal and bimanual examination it could not be differentiated from the uterus. The lower pole was felt through the anterior and left lateral fornices, and a nodule through the right fornix.

On opening the abdomen a considerable quantity of free fluid was evacuated. The peritoneum and intestines were studded with nodules evidently malignant in character. The left Fallopian tube was much enlarged, and formed the swelling in the left side of the abdomen. It was hard and nodular, and adherent to the intestines and to the ovary, which was also covered with secondary nodules. On the right side the ovary was the seat of a small cystic tumour about the size of a fœtal head. It also was covered with nodular growths. The tubes and ovaries on both sides were removed, but it was impossible to deal with the secondary deposits.

After the operation the patient had a great deal of sickness, and she died eleven days after operation. Unfortunately no post-mortem examination could be made.

Specimen.—The specimen consists of the left tube and ovary. The tube is enlarged towards its ampullary and infundibular regions. The uterine end is of normal size, and on transverse section its lumen is not dilated. Beyond the isthmus it rapidly expands, the lumen in this region being dilated and filled with mucus which has coagulated with the formalin. Beyond this dilated portion the tube forms a solid tumour mass which retains a convoluted shape, but in which the fimbriated extremity cannot be distinguished.



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The whole mass measures 4 inches in length and 7 inches in circumference.

In the peritoneum on the surface are numerous dilated veins and several small nodules. Towards the outer extremity there is a fungating part of the growth which has broken through the peritoneal surface. On section of this part of the tube all trace of lumen is lost. The cut surface is of a yellowish-white colour, and of brain-like consistence and appearance. In it are several hæmorrhagic areas, and in places the tissue is softened and broken down.

The ovary is slightly enlarged and is covered with small nodules, which on section have the same appearance as the growth in the tube.

The ovarian tumour from the right side has the ordinary characters of a cyst-adenoma, but on the surface are many small secondary nodules apparently implanted there.

So far as examination of the parts removed can show, the primary growth seems to be in the tube; but it is impossible to be absolutely definite on this point, as in the absence of a post-mortem examination the presence of a tumour elsewhere in the peritoneal cavity cannot be excluded. There was, however, no evidence of such at the time of operation.

Microscopic Examination.—Sections of the isthmus of the tube show no disease, nor do these taken from the dilated portion immediately beyond this. In this region there are, however, secondary nodules of growth in the peritoneal surface. In the outer part of the tube, which has been converted into a solid mass, the typical structure is seen. The great part of this growth is made up of malignant cells, and in most of the areas examined the perivascular arrangement of these cells is evident, with areas of necrosis and hæmorrhage interspersed (Plate III., Fig. 1). The appearance of the vessels differs from that described in the perithelioma of the uterus. The endothelial lining of the vessels is intact, and

outside it is a clear zone of material which stains homogeneously and only very feebly, resembling hyaline. Beyond this hyaline zone the tumour cells are arranged radially to the vessel lumen. The cells are large and irregular in outline, with central darkly staining nucleus (Plate III., Fig. 2). In parts the cells are seen in solid clumps with no vessel in the centre, but this is probably in most cases due to the direction of section. In the secondary deposits on the surface of tube, ovary, and the right-sided ovarian cyst, the perivascular arrangement of the cells is not seen. They are in solid masses, but have the same characters as those forming the tubal tumour.

Of these two cases the tubal tumour is evidently the more malignant in character. A study of the literature shows that the degree of malignancy varies greatly in different cases, many apparently being on the border-line between innocence and malignancy.

Lately there has been a tendency to regard peritheliomas as sarcomas, with necrosis and persistence of the tumour cells in the region of the vessels. Roussy and Amenille[1] support this view after a careful study of many specimens of all the literature. To this paper the reader is referred for a full discussion of the subject. In his recent paper on tumours of the uterus, Leith (*Journ. of Obstet. and Gyn. of the Brit. Emp.*, April and May 1911) describes specimens of sarcoma with this perivascular arrangement, and discusses their possible endothelial origin. Cases in the uterus have also been described by Doran and Lockyer.[2]

The President thanked Dr Watson for bringing his paper before the Society, but was sorry he had not known previously. He (Dr Haultain) had shown a tumour a few months ago

[1] *Bull de l'Assoc. Trans. pour l'Etude du Cancer*, tome iv., No. 3.
[2] *Journ. of Obstet. and Gyn. of Brit. Emp.*, 1908.

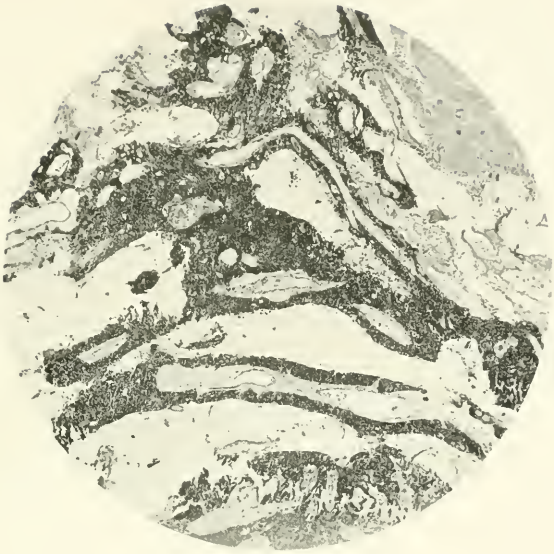

FIG. 1.

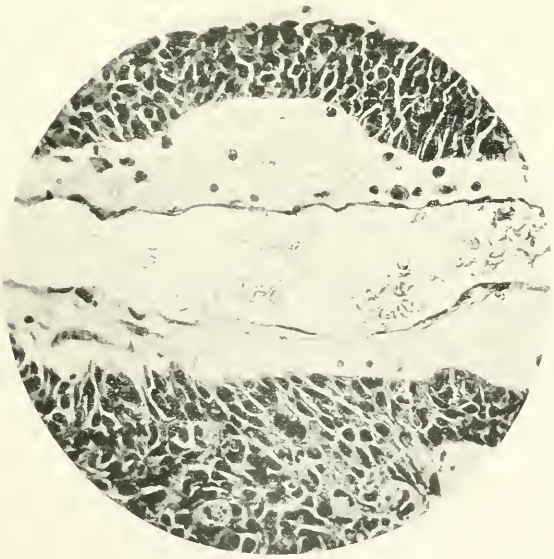

FIG. 2.

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which turned out to be exactly the same. He was sorry to say that the malignancy in that case had been very great. He removed the entire uterus and a considerable part of the vagina, but already there was a return of the growth, which was progressing extremely rapidly.

Dr Berry Hart was much interested in the paper. They were indebted to the gynæcologists for much information. Some years ago if a tumour was solid it was a fibroid, and no idea of its being anything else was entertained. He remembered soon after he began to practise that Dr Mathews Duncan said he would show him a big malignant fibroid. He (Dr Berry Hart), thinking that Dr Duncan was just "putting it on," said it was big for malignant disease. That was the first case!

Description of Figures.

Plate

I.—Fig. 1.—Perithelioma of Uterus. The tumour has a wide origin from the uterine wall, and is causing thinning of it by invasion. The greater part is necrotic.

Fig. 2.—Typical appearance of Tumour. Large areas of necrosis with islands consisting of a vessel surrounded by tumour cells.

II.—High-power view of Vessel showing Perivascular Arrangement of Cells.

III.—Fig. 1.—Perithelioma of Tube. Note the hyaline zone between the endothelial lining of the vessels and the tumour cells.

Fig. 2.—High-power view of Vessel and Tumour Cells.

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